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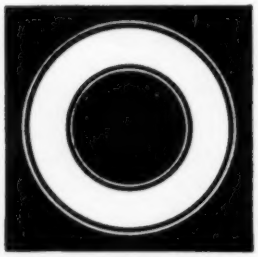

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CONTENTS

THE WHOLE OF THE LITERARY MATTER IN THE LANCET IS COPYRIGHT

ADDRESSES AND ORIGINAL ARTICLES

Surgical Treatment of Angina Pectoris and Allied Conditions. By DANIEL T. DAVIES, F.R.C.P., H. E. MANSELL, F.R.C.P., and L. O'SHAUGHNESSY, F.R.C.S. With the co-operation of VISCOUNT DAWSON OF PENN, P.R.C.P. (*illus.*) (*To be concluded*) 1

A New Syndrome Apparently due to Over-activity of the Posterior Pituitary. By E. IDRIS JONES, M.R.C.P. (*illus.*) .. 11

Clinical Hyperfunction of the Posterior Lobe of the Pituitary. By R. L. NOBLE, M.D., H. RINDERKNECHT, and P. C. WILLIAMS, B.Sc. (*illus.*) .. 13

Electrosurgical Obliteration of the Gall-bladder without Drainage. By Prof. MAX THOREK (*illus.*) 15

Vitamin-C Output in Diet Treatment of Rheumatoid Arthritis. By D. C. HARE, F.R.C.P., and E. C. P. WILLIAMS, M.B. (*illus.*) .. 20

Prevalence of Sonne Enteritis. By L. H. D. THORNTON, M.R.C.S., and E. M. DARMADY, M.R.C.P. 25

CLINICAL AND LABORATORY NOTES

Reciprocal Rhythm. By C. W. CURTIS BAIN, M.R.C.P. (*illus.*) 26

A Modification in the Treatment of Extensive Varicose Veins. By D. L. STEVENSON, F.R.C.S., and R. M. WALKER, F.R.C.S. 27

MEDICAL SOCIETIES

North of England Obstetrical and Gynæcological Society: Sulphanilamide in Obstetrics 28

REVIEWS AND NOTICES OF BOOKS

Developmental Abnormalities of the Eye. By Ida Mann, F.R.C.S. 30

Diseases of the Blood and Atlas of Hæmatology. By Prof. R. R. Kracke, M.D., and H. E. Garver 30

Story of a Great Hospital: The Royal Infirmary of Edinburgh, 1729–1929. By A. Logan Turner, F.R.C.S. 30

Trauma and Disease. Edited by L. Brahdy, M.D., and S. Kahn, M.D. 31

Thorpe's Dictionary of Applied Chemistry. Vol. I. By Prof. J. F. Thorpe, F.R.S., and M. A. Whiteley, D.Sc. 32

Malaria and Ankylostomiasis in the Pregnant Woman. By G. A. W. Wickramasuriya, F.R.C.P. 32

Pediatric Urology. By Prof. M. F. Campbell, M.D., and J. D. Lyttle, M.D. 32

LEADING ARTICLES

ANTISERA IN RELATION TO CANCER 33

ÆTIOLOGY OF RENAL CALCULI 33

LATENT SQUINT AND THE AIRMAN 34

LOCAL TREATMENT OF THE COMMON COLD 35

ANNOTATIONS

A New Pituitary Syndrome 36
The Dangers of Thorotrast 36
Precordial Leads in Electrocardiography 37
The Mental Effects of Altitude 37
Embolism under Observation 38
Peritoneoscopy 38
The Anæmia of Iron Deficiency 39
Euphyllin in Cardiac Disease 39
An Inert Excretion Product of Progesterone 39
Pentothal Sodium 40
Who Pays the Doctor Called to an Accident? 40
Health of the School-child 41
Bismuth Poisoning 41
Medical Oxford and the Dominions 41

SPECIAL ARTICLES

Safety Factor of Designated and Other Types of Liquid Milk. By W. G. SAVAGE, M.D. 42

Medicine and the Law: Romance 43

United States of America: Future of Voluntary Hospitals 44

Bucharest: Public Health Services 45

Croydon Typhoid Inquiry (*continued*) 54

Health of the Navy 57

THE LANCET 100 Years Ago 29

PANEL AND CONTRACT PRACTICE

Payment of Anæsthetists.. 46

PUBLIC HEALTH

Confidential Reports—Youthful Offenders—A List of Sanatoria 47

Infectious Disease in England and Wales 59

GRAINS AND SCRUPLES

From a Bacteriological Back-number (I) 48

PARLIAMENT

Rise in the Cost of Living .. 49

Notes on Current Topics: Coal Bill and Miners' Welfare 49

Question Time: Prevention of Silicosis — Salaried Midwives — Asbestosis Certificates—Health Insurance and Women's Benefits — Disease Carriers'—Health Insurance and Ophthalmic Benefit—Local Government and Public Health in Scotland 50

CORRESPONDENCE

Carcinoma of the Stomach (Dr. Charles Miller) 52

Treatment of Shock (Mr. J. W. Riddoch, F.R.C.S.) 52

Aplastic and Achrestic Anæmias (Drs. J. F. Wilkinson and M. C. G. Israëls) 53

Melanosis Coli (Dr. H. L. Bockus) 53

Wanted Specimens of Normal Semen (Mr. K. M. Walker, F.R.C.S.) 54

Child Discipline (Mr. A. S. Neill) 54

OBITUARY

George Alfred Buckmaster, F.R.C.S. (*with portrait*) .. 58

Charles Sanders, M.B. (*with portrait*) 58

MEDICAL NEWS

University of Cambridge .. 59
University of London 59
Society of Apothecaries of London 59
National University of Ireland 59
Royal Society of Medicine of Budapest 59
University of Bristol 60
Regional Coördination of Voluntary Hospitals 60
The Oxford and Bath Congresses on Rheumatism and Hydrology 60
Mental After-Care Association 10

NOTES, COMMENTS, AND ABSTRACTS

The Social Care of the Epileptic. By RALPH H. CROWLEY, M.D..... . 61
The Dionne Quintuplets . 62
Geodesy and Ciliary Action 62
Rest in Bed 63
New Preparations: Cow and Gate (Vitamin D) Malted Milk 63
An Article Wanted 63

Appointments 60
Births, Marriages, Deaths .. 60
Vacancies 64
Medical Diary 64

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ADDRESSES AND ORIGINAL ARTICLES

SURGICAL TREATMENT OF ANGINA PECTORIS
AND ALLIED CONDITIONS

BY DANIEL T. DAVIES, M.D.Wales, F.R.C.P.Lond.

H. E. MANSELL, B.M. Oxon., F.R.C.P. Lond.

AND

LAURENCE O'SHAUGHNESSY, F.R.C.S. Eng.

With the coöperation of

VISCOUNT DAWSON OF PENN, P.C., M.D., B.Sc., P.R.C.P. Lond.

(*From the Cardiovascular Clinic, Lambeth Hospital, London County Council*)

THE experimental basis and technique of cardio-omentopexy was described a year ago in a special lecture at the Royal College of Surgeons of England which was introduced by Lord Dawson of Penn, honorary consultant to the Lambeth Cardiovascular Clinic, and published in this journal. The present paper contains an account of the further experiences of the clinic with cardio-omentopexy and other surgical means of supplementing a deficient coronary circulation.

In the Registrar-General's report for 1935 12,317 deaths are recorded from angina pectoris and coronary disease, and it seems probable that cardiac ischæmia played an important part in at least some of the enormous total of 70,504 deaths from myocardial disease. The total figure for carcinoma is 66,000.

The relation of coronary disease to sudden death has long been realised. Leyden, writing in the eighties, recognised the clear relation between degenerative changes in the myocardium and sclerosis of the coronary tree. Huchard agreed with Leyden, but by 1915 Sir Clifford Allbutt, while not denying the occasional existence of gross coronary obstruction as a cause of death, regarded angina as a symptom not of cardiac ischæmia but of some undefined type of nervous degeneration in the aortic plexus. To-day by common consent opinion has returned to the older view. On the clinical side Levine, in his clear and concise monograph, has grouped under the general heading of Angina Pectoris all the organic causes of cardiac ischæmia—coronary thrombosis, coronary sclerosis, and syphilitic aortitis—and he is emphatic that in fatal angina structural change is always present both in the myocardium and in the coronary tree. It is gradually being recognised that "chronic myocarditis" is a comparatively rare disease apart from cases of syphilitic or rheumatic origin, and that the greater proportion of cases so labelled, if carefully examined post mortem, show evidence of coronary disease.

Büchner, Weber, and Haager (1935) have published a careful record of 43 fatal cases of cardiac ischæmia in which the clinical findings are correlated with the post-mortem appearances both macroscopic and microscopic. They describe coronary thrombosis in an otherwise normal coronary tree, thrombosis complicating atheroma of the coronary arteries, hypertensive heart failure with gross atheroma of the tree but no gross obstruction, and syphilitic aortitis in which only the orifices of the vessels were affected. In every case the electrocardiograms were characteristic of cardiac ischæmia, and at autopsy careful examination of the heart revealed pathological changes in the myocardium. After obstruction of a main vessel, gross evidence of an old or recent infarct was invariably present; and where no actual block of the circuit was found but merely a general impairment of its lumen, scattered areas of recent necrosis or old fibrosis could always be detected in serial sections.

Under some conditions coronary occlusion is a fatal event, but the organism is often in a position to effect a remarkable degree of compensation; even successive attacks of coronary thrombosis may be survived and an astonishing degree of normal activity may be regained in the interval, sometimes years in extent, between the initial and the second attack. The mechanisms by which this natural compensation may be brought about have already been considered in previous papers. Although it is quite possible that there may be some subtle mechanism of which we know nothing, there are only two methods for the efficacy of which we have experimental and clinical evidence. One of these is anastomosis between the right and left coronary arteries, and the other is the process by which the heart may supplement its blood-supply from the parietes. The first mechanism is clearly of no avail when a diffuse sclerosis affects the entire coronary tree, and the second is only available if partial or complete destruction of the epicardium takes place so that adhesions may form between the heart and the parietal pericardium. After a coronary thrombosis only a small proportion of patients form adhesions between their infarct and the parietal pericardium, but this mode of compensation is important because it alone can be initiated or supplemented by surgery.

In a previous paper examples of the beneficial association of pericarditis with coronary disease were reviewed—Sternberg's classical case of "pericarditis epistenocardica," the histories cited by Mönckeberg, and also Sir Clifford Allbutt's doctor patient who returned to active practice after a severe attack of angina complicated by pericarditis. Chance has provided us with an excellent example of natural compensation in angina pectoris.

> The father of one of our operated patients is now aged 68. For several years he suffered from angina for which he received the usual medical treatment. Three years ago he was suddenly seized by a more violent attack of pain in his chest than he had previously experienced, and in a state of collapse he was removed to hospital, where he passed some days in a critical state and grave doubts of his ultimate recovery were expressed. However, he recovered and after some weeks in bed returned home. He gradually resumed general activity and was amazed to find that his angina had disappeared. He remains free from pain and our interpretation of his recovery is that he suffered an attack of coronary thrombosis and adhesions formed with the parietes.

Moritz demonstrated by injection studies the vascularity of pericardial adhesions, and similar results have been obtained by one of us (H. E. M.) in the pathological department at Lambeth. In cases of pericarditis following coronary thrombosis it was possible to demonstrate vascular continuity between the coronary tree and the vessels of the parietal pericardium, the inferior sternopericardial ligament, and the diaphragm. It may be mentioned that the vascularity of the structures surrounding the heart in human beings appears to be much greater than in experimental animals.

A review of the literature of coronary disease shows that methods of initiating or supplementing the collateral blood-supply to the heart are engaging the attention of physicians both in America and in Germany. Hochrein in his latest monograph (1937) states that "we have learnt to recognise that peri-

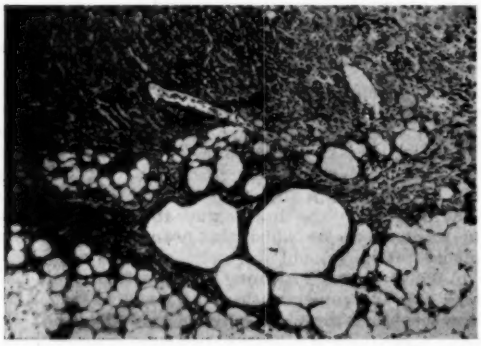

FIG. 1.—Section through junction between myocardium (above) and omentum (below) a week after operation. A primitive vessel is growing from the omentum into the heart muscle. (× 60.)

carditis is not a complication of coronary thrombosis but a natural attempt at healing—its presence is to be welcomed rather than deplored," and he goes on to discuss the theoretical possibility of encouraging the formation of pericardial adhesions when nature has failed to provide them. This theoretical possibility has been carried into practice in some of our cases with apparently beneficial results.

Experimental Basis of Cardio-omentopexy and Allied Operations

Experimental work on various methods of augmenting the blood-supply to the heart has been in progress at the Buckston Browne farm of the Royal College of Surgeons of England since April, 1933. The results previously obtained have been recorded in various publications, and a further paper containing details of modifications in technique is in preparation.

In animal experiments we have shown that an omental graft attached to the heart is compatible with the highest degree of physical exertion. One of our greyhounds completed a full course (525 yards) without distress after his recovery from two operations—at the first the descending branch of the left coronary artery was tied and at the second the graft was applied.

We have also shown that vascular connexions rapidly form between the graft and the myocardium, whether it is attached to normal muscle, to the site of a recent infarct, or to the site of an old fibrous infarct; this is important because these are the three conditions with which one might be confronted at operation. New vessels may be observed microscopically at the end of a week (Fig. 1), and at the end of three weeks complete injection of the coronary tree may be carried out from the graft with a suspension of indian ink. As time passes the vascular connexion increases and these injections may be made with barium paste (Fig. 2). The oldest specimen examined—that of a greyhound killed eighteen months after operation—showed that there was a marked increase in the normal mediastinal vessels and that these too had made connexions with the graft and with the myocardium. It is a fundamental point that cardio-omentopexy brings a new blood-supply to the heart from the abdominal aorta and also supplements the normal collaterals in the mediastinum and gives them access to the ischæmic heart. It is also important to realise that the graft forms direct connexions with the myocardium at its point of attachment and that its vessels anastomose with branches of the coronary tree, and therefore blood brought by the graft may be distributed to all parts of the heart. In other words, exact placing of the graft in relation to any localised area of ischæmia which it is desired to influence is not essential.

Lezius (1937) has shown that the lung can form extensive vascular adhesions with the heart when the fibrous pericardium is removed; he found that under these conditions he could ligature the descending branch of the left coronary artery without causing the usual infarct. In cardio-omentopexy the lung often adheres to the graft and probably assists in revascularisation, but we have so far only used cardio-pneumonopexy when the usual operation was impossible.

In the most recent of our laboratory experiments we have shown that the production of intrapericardial adhesions by the use of a special preparation—Aleuronat—provided us with an additional and simple means of revascularisation. In this way a route is afforded for revascularisation from the mediastinal vessels, but the method is inferior to cardio-omentopexy in that reinforcement of these vessels does not occur.

In our earlier experiments and operations the omental graft was always sutured to the heart; more recently we have been able to obtain equally firm attachment of the graft by suturing it to the pericardium only and applying aleuronat paste between the graft and the heart. We have also been able to demonstrate that the vascular continuity under these conditions is of the same order as when sutures are employed. This is technically important, for our experience has shown the danger of placing sutures in a friable and degenerate heart.

The Operations

Four types of operation were used in this series:

Cardio-omentopexy (Fig. 3) in which a left transpleural exposure of the heart is used, the pericardium is opened and a pedicled graft of the great omentum is brought through the diaphragm and attached to the heart.

Transpleural pericardiotomy in which the pericardium is opened after a left transpleural exposure, aleuronat paste is inserted and the pericardium is closed.

Extrapleural pericardiotomy in which the seventh costal cartilage and part of the xiphoid process are resected in Larrey's angle, the pericardium is opened and the sac treated with aleuronat.

Cardio-pneumonopexy (Lezius) in which after a left transpleural approach the pericardium is opened and a portion of lung is attached to the heart.

The Cases

The series consists of twenty cases. Patients were selected for operation who showed unequivocal evidence of cardiac ischæmia and who had failed to benefit by medical treatment. The majority were suffering from angina of effort. The aim of the operation in every case was to supplement the

coronary circulation from without. The actual technique was modified to suit the individual patient and varied from complete cardio-omentopexy to simple pericardiotomy for insertion of the irritant aleuronat to encourage pericardial adhesions.

The cases will be considered in two groups—the first and larger group consisting of patients suffering from angina pectoris, the second and smaller group consisting of patients with other symptoms of cardiac ischæmia.

GROUP I: ANGINA PECTORIS

Of the fifteen members of this group (Table I) some were bedridden, others were in hospital, and the rest with one exception suffered from angina of effort of such degree as to curtial their ordinary activity. The exception is Case 13, in which the pain was not severe and was due to his previous coronary thrombosis, but in view of his age we considered operation justifiable.

CASE 1

A cosmopolitan of varying occupations, aged 65, was transferred to Lambeth Hospital on July 28th, 1936, from St. Stephen's Hospital (L.C.C.), where he had been admitted on July 7th after an attack of severe angina on the previous day. The pain had been most severe, lasting for four hours without relief from amyl nitrite and accompanied by much dyspnœa. He had had eight attacks during the previous four years, and from the first attack had limited his activities as far as possible. He was not normally breathless on exertion. He had had severe insomnia for the past six or eight months. He was a heavy smoker until three years ago. He gave a history of malaria and three laparotomies—for appendicitis, adhesions, and gastro-enterostomy. A paternal uncle had "dropped dead of angina."

Physical *examination* showed a grey-haired, well-nourished man with somewhat protruding eyes. His tongue was clean. There was no enlargement of the thyroid but slight tremor of the fingers. The radial artery was slightly tortuous but not thickened. The heart was not enlarged, the sounds were normal, and there was a soft apical systolic murmur. The blood pressure was 116 mm. systolic and 72 diastolic. The lungs were emphysematous. The abdomen showed three well-healed scars. The right pupil was larger than the left and the knee-jerks and ankle-jerks were not elicited. The urine was normal and the blood-urea 32 mg. per 100 c.cm. The Wassermann reaction was negative. X ray examination showed a large heart shadow. The basal metabolic rate was −40 per cent. The electrocardiogram showed the S–T interval raised and convex downwards in leads II and III.

Cardio-omentopexy was performed on August 17th. The heart was found to be free of pericardial adhesions. There was no difficulty in obtaining omentum in spite of three previous laparotomies.

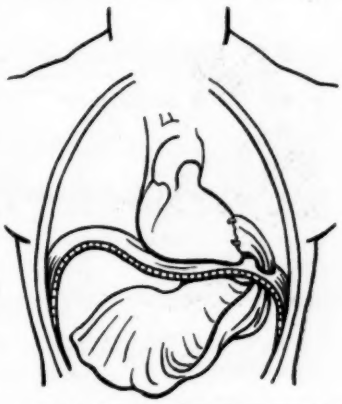

FIG. 3.—Diagram illustrating cardio-omentopexy in man.

At the end of operation the upper left intercostal nerves were injected with 1 per cent. novocain. The pulse-rate was 96. Except for an immediate mild bronchitis recovery was uneventful. He was allowed up four weeks later and took his own discharge on Dec. 10th, the blood pressure being 160 mm. systolic and 90 diastolic. There were no further attacks of angina. Movement of the left diaphragm was seen for the first time 3½ months after operation. Serial electrocardiograms showed a gradual emergence of a Q wave in lead III.

Since leaving hospital he has been able to lead a life of normal activity without symptoms.

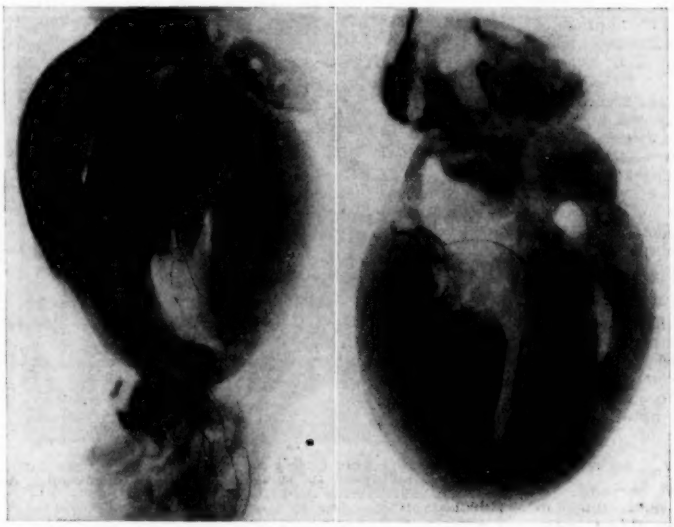

FIG. 2a.—Greyhound's heart injected with barium paste through the graft, eighteen months after cardio-omentopexy. Left side with graft attached.

FIG 2b.—Right side of the same heart.

CASE 2

A male, aged 71, who had retired from the Burma Police thirty years previously, was admitted to Lambeth Hospital from Dr. Dean Pollard of Bedford on July 31st, 1936, complaining of attacks of pain, beginning at the base of the neck and spreading over the heart and down the left arm. The first attack had occurred suddenly during exertion three years previously. They had become increasingly frequent—now almost daily—but not more severe. The pain was always related to exertion, emotion or, during sleep, to nightmare, and was relieved by nitroglycerin. He was not breathless on exertion between the attacks. He gave a history of appendicectomy in 1912.

Physical *examination* showed a well-nourished man of rather plethoric facies. His remaining teeth were septic. The radial arteries were tortuous and thickened. The size of the heart was masked by emphysema. The sounds were very distant but not spaced, and there was a soft systolic murmur at the apex. The blood pressure was 205 mm. systolic and 105 diastolic. Nothing abnormal was found in the lungs, abdomen, and central nervous system. The urine was normal and the blood-urea 44 mg. per 100 c.cm. The Wassermann

TABLE I—CASES OF ANGINA PECTORIS

Case	Age.	Sex.	Diagnosis.	Operation.	Date.	Result.
1	65	M.	Angina of effort.	Cardio-omentopexy by suture only.	Aug. 17, 1936	Symptom-free.
2	72	M.	" "		Sept. 25 "	Died of uræmia three months later.
4	65	F.	Angina, hypertension, bundle-branch block.	Cardio-omentopexy by suture with insertion of aleuronat.	Oct. 16 "	Symptom-free.
5	60	M.	Angina of effort.		Jan. 22, 1937	" "
7	67	F.	" "	..	Feb. 26 "	Died on table.
8	56	M.	" "	Pericardiotomy for insertion of aleuronat.	Mar. 5 "	Symptom-free, at work.
9	50	M.	Coronary thrombosis, angina of effort.	Cardio-omentopexy with aleuronat only.	Mar. 12 "	" "
10	41	M.	Angina of effort.		April 16 "	Much improved, at work.
11	56	M.	" "		April 23 "	Improved, at work.
13	43	M.	Coronary thrombosis.		May 7 "	Symptom-free, at work.
14	56	M.	Angina of effort, auricular fibrillation.	Transpleural pericardiotomy for insertion of aleuronat.	May 28 "	Died two months later.
15	65	F.	Coronary thrombosis, angina of effort.	Cardio-omentopexy by suture only.	June 4 "	Much improved, discharged to convalescent home.
17	65	M.	Angina of effort.	Cardio-omentopexy with aleuronat only.	July 2 "	Died two months later.
18	62	F.	" "		July 2 "	" "
19	55	M.	" "	Pericardiotomy for insertion of aleuronat.	Aug. 25 "	Discharged from hospital, in statu quo.

reaction was negative. X ray examination showed that the heart was not enlarged. The electrocardiogram showed left axis-deviation, with inversion of the T waves in lead I. The S–T interval was depressed in lead I and raised in lead III.

While *in hospital* he had two attacks of angina: a mild attack in our presence while walking too fast, and a severe attack lasting six or seven minutes while straining at stool. On August 17th he developed acute tonsillitis and was sent home to recuperate on the 26th. He was readmitted on Sept. 23rd; he had a severe attack lasting twenty minutes on the station, after climbing a flight of 24 steps twice, and had to take two tablets of nitroglycerin before being able to proceed. On examination he looked fit and the throat was clear. The blood pressure was 208 mm. systolic and 118 diastolic. There were no basal râles and the urine was normal. The electrocardiogram showed no change.

Cardio-omentopexy was performed on Sept. 25th. The pericardium was tense, and the heart enlarged but free of adhesions. The pulse-rate at the end of operation was 72, and the blood pressure two hours later was 154 mm. systolic and 92 diastolic. The electrocardiogram showed no change.

He developed purulent bronchitis which lasted a week. On Oct. 4th he had two attacks of angina following visiting hours, the second needing an injection of morphia. There was a slight attack two days later. On Oct. 12th he had a severe attack (after being allowed up for two hours by mistake), only partially relieved by nitroglycerin and morphia and leaving numbness of the left arm. He subsequently became subject to frequent attacks of pain, occurring day and night and not related to exertion. The pain was relieved by nitroglycerin but not by an injection of sterile water. Attacks did not occur when his attention was occupied. He began to get up at the end of four weeks and was discharged home on Nov. 1st, showing no ill effects from a long car journey. His wife wrote on Nov. 30th: "He is immensely improved both mentally and physically, and I think that he improves slowly all the time. If it were not for occasional pains, he would be in perfect health." During the following week there was a return of severe pain and insomnia. The pain soon eased off, to be followed by breathlessness which became more and more distressing until Dec. 19th, when he was *readmitted* to Lambeth Hospital.

On examination he looked haggard and desperately ill. There was Cheyne-Stokes breathing, but in spite of the violence of the hyperpnœic phase he had no angina. The pulse was of fair volume and rate 100. The heart sounds were of very poor quality and there was gallop rhythm. The blood pressure was 140 mm. systolic and 100 diastolic. Râles were audible at the left base. There was no œdema but the urine was loaded with albumin. The tongue was dry and coated. The electrocardiogram showed much increase in voltage of the S waves in lead III and changes in the S–T interval in leads I and III. A diagnosis of uræmia was made. The blood-urea proved to be 116 mg. per 100 c.cm. and the blood calcium 10·4 mg. He improved

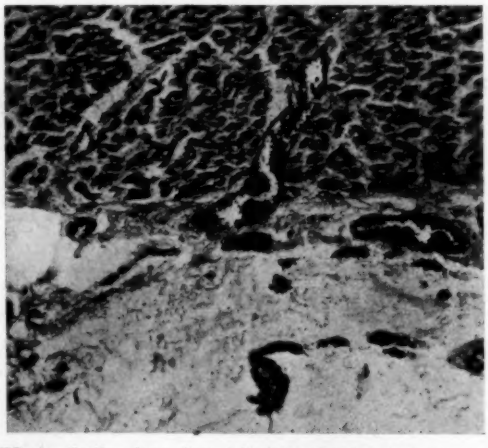

FIG. 4.—Section from Case 2 through junction between myocardium and omentum three months after operation. A vessel is seen entering the myocardium.

for a few days with treatment but changed suddenly for the worse on Dec. 27th and died that night.

Post-mortem examination showed the operation wound to be firmly healed and there had been complete restitution of the thoracic cage. The graft was firmly attached to the heart, which was removed for injection. The right pleural cavity contained free fluid in excess but the lung was normal. The lower lobe of the left lung was collapsed. The abdomen contained an excess of free fluid, but the viscera, including the colon, were normal, and the graft could be seen in place passing through the diaphragm. The kidneys showed chronic interstitial nephritis and there was advanced atheroma of the abdominal aorta. Injection of the graft with a watery suspension of indian ink was carried out from the abdominal aorta, after removal from the body. A pressure of 100 mm. of mercury was used, and the technique was identical with that employed in the experimental series. A very profuse injection of the parietal pericardium was obtained, and the ink eventually reached the chambers of the heart. The specimen was fixed in formalin, and on section it was possible to see ink lying in the myocardium. Stained sections showed new vessels running from the omentum into the myocardium (Fig. 4). There was diffuse sclerosis of the coronary tree, the lumen being obliterated at one point on the left descending branch, but there was no actual infarction of the myocardium.

CASE 4

A missionary's widow, aged 65, was admitted to Lambeth Hospital from Dr. Eyne of Hove on Oct. 1st, 1936, with a history of angina of twelve years' duration. It had formerly been relieved by amyl nitrite but now needed Omnopon. Attacks were often occurring at night, or in the early morning if she had become overtired the evening before. She had been practically confined to bed for eighteen months past and was unable now to wash herself. She was also liable to attacks of rapid and irregular pulse, lasting about half an hour and causing considerable distress at the time. She also complained of headache and a bursting feeling in the head. She had led an exceptionally adventurous and strenuous life, often at high altitudes. She had had pleurisy several times, and pneumonia eight years ago. There is a family history of pulmonary tuberculosis. A sister died of a stroke and another has angina.

On *examination* she was obese. The teeth were well stopped, the throat clear. The apex-beat was in the 5th intercostal space in the anterior axillary line. The mitral first sound was very low and the aortic second sound accentuated and ringing. There was a very soft to-and-fro murmur at the apex and in the aortic area. The blood pressure was 247 mm. systolic and 117 diastolic. Nothing abnormal was found in the chest and abdomen. The knee-jerks and ankle-jerks were not obtained. There was no œdema and the urine was normal. The Wassermann reaction was negative. X ray examination showed a large heart. The electrocardiogram showed a right (old terminology) bundle-branch block, with very large amplitude of the Q R S complexes in leads I and III. (The bundle block was known to have been present in 1930.) She was treated for fourteen days with a starch-free diet, general massage, and sedative drugs.

Cardio-omentopexy was performed on Oct. 16th. The inferior sternopericardial ligament was fatty and well marked. A large fatty graft was secured and sutured to the heart, which was free of adhesions, aleuronat in addition being inserted. At the end of operation the pulse-rate was 69 and the blood pressure 170 mm. systolic and 90 diastolic. Recovery was interrupted by a paroxysm of auricular fibrillation, which began 30 hours afterwards and ceased spontaneously 9½ hours later. It was accompanied at the onset by severe angina. There was a moderate rise of evening temperature for four days. Two anginal attacks occurred in the first fortnight and none subsequently. General massage was restarted after five weeks and she was allowed to wash herself. In six weeks the blood pressure had returned to 240 mm. systolic and 112 diastolic. On Christmas Day, ten weeks after operation, she was up and about the ward for two hours without ill effects. During January she had some further attacks of paroxysmal fibrillation accompanied by pain. They ceased as soon as she was put on quinidine. She was transferred to St. Benedict's Hospital for convalescence on Feb. 3rd, sixteen weeks after operation. The blood pressure was 230 mm. systolic and 116 diastolic; the electrocardiogram showed no change and there were still signs of collapse at the left base.

She was discharged from St. Benedict's Hospital on March 18th and has gradually regained her activity. There have been no more attacks of angina. There was a brief return of auricular fibrillation in June when she had been overdoing things, but it responded at once to quinidine. Eight months after operation X ray examination

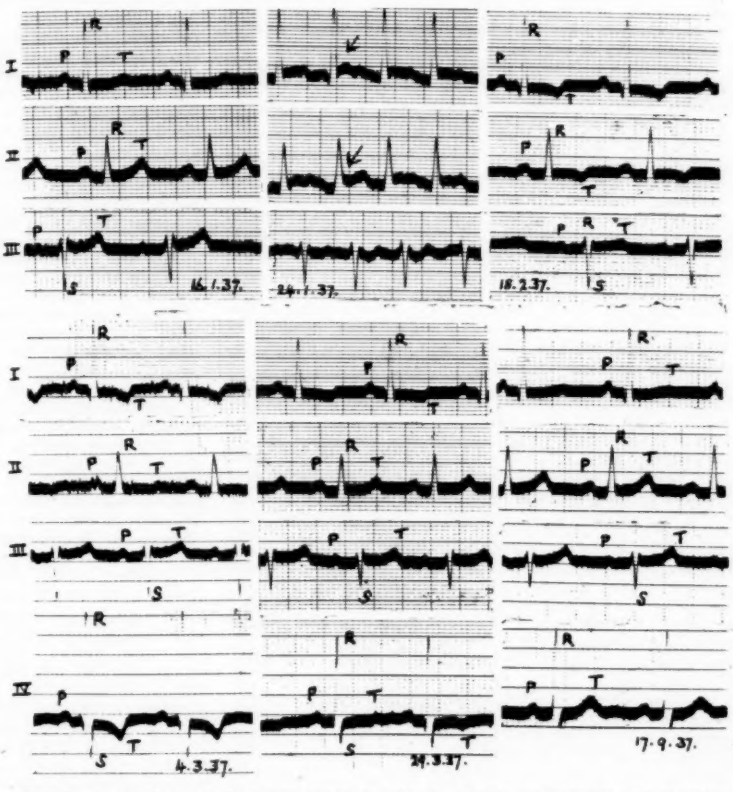

FIG. 5.—Serial electrocardiograms taken in Case 5. Cardio-omentopexy with insertion of aleuronat was performed on Jan. 22nd.

showed the left diaphragm moving synchronously with the right, and there were no longer signs of collapse at the left base. She is now leading a life of activity normal for her age without any pain.

CASE 5

An engineer, aged 60, was admitted to Lambeth Hospital on Jan. 12th, 1937, with a four years' history of angina pectoris. Attacks had gradually increased in frequency and during the past few months had made his life unbearable. They had occurred occasionally in bed. Pain was relieved by nitroglycerin. He had led a very active life, chiefly in the tropics for the last thirty years. He was a strong man, well known for weight-lifting and wrestling, a big spirit-drinker and heavy smoker. There was no family history of angina.

Physical *examination* showed a well-developed man of height 5 ft. 8½ in. and weight 13½ st. There was no evident arterio-sclerosis. The heart was not enlarged, the mitral first sound was accentuated and low-pitched, the aortic second sound clear, and there were no murmurs. Nothing abnormal was found in the chest, abdomen, and central nervous system. There was a small left inguinal hernia. The urine was normal and the blood-urea 32 mg. per 100 c.cm. The Wassermann reaction was negative. X ray examination showed no enlargement of the heart. The electrocardiogram showed left axis-deviation and low T waves in lead I.

Cardio-omentopexy was performed on Jan. 22nd. The omentum was easily obtained, but some difficulty was met in getting enough without bringing the stomach with it. The heart was not enlarged and was free of adhesions. Aleuronat was applied and the graft was sutured to the heart and pericardium. The pulse-rate at the end of operation was 108 and the blood pressure 140 mm. systolic and 85 diastolic. He developed mild purulent bronchitis and fever lasting a fortnight. During the first week he had several paroxysms of auricular fibrillation, but these were readily controlled by quinidine. During the second week he developed signs and X ray evidence of a small left pleural effusion. At the end of a fortnight he had an attack of angina, and in less than a month he was getting attacks as often as before the operation. They were relieved by nitroglycerin. He was allowed up at the end of four weeks, and was transferred to Sidcup for convalescence on March 30th, his blood pressure being 156 mm. systolic and 96 diastolic. Serial electrocardiograms (Fig. 5) showed inversion of the T waves in leads I, II, and IV, most marked between the fourth and sixth weeks after operation.

Attacks of pain became gradually less frequent and the consumption of nitroglycerin tablets less, but it was seven months before he reported himself symptom-free. Before operation five stairs were all he could manage. He now climbs sixty to his room without pain or distress. In November he successfully underwent radical herniotomy under general anæsthesia.

CASE 7

A widow, aged 67, was admitted to Lambeth Hospital on Feb. 5th, 1937, at her own request, complaining of angina pectoris of two years' duration. The attacks were frequent and severe, and she could not even put on her shoes without inducing pain. Nitroglycerin gave her short-lived relief. Against her family's wishes she desired operation, even if it were to prove fatal. Ten years ago she had been treated in Bethnal Green Hospital for hypertension. Two years ago she had a stroke with left hemiplegia. There was a history of pleurisy as a young woman. Her mother died of "aneurysm of the heart."

Physical *examination* showed a well-nourished, grey-haired woman. The brachial arteries were full and elastic. The apex-beat was in the 5th intercostal space, 4½ in. out. The cardiac impulse was heaving. The mitral first sound was low-pitched and the sounds rather spaced. A systolic murmur was audible in all areas and maximal in the pulmonary area. The blood pressure was 200 mm. systolic and 118 diastolic. There were râles at the bases. The abdomen was normal. The right pupil was smaller than the left and did not react, and the ankle-jerks were not obtained. There was very slight œdema over the tibiæ. The urine and kidney function tests were normal and the blood-urea 36 mg. per 100 c.cm. The Wassermann reaction was negative. X ray examination showed the right diaphragm raised but moving normally. The heart was not enlarged. The electrocardiogram showed well-marked Q waves and flat T waves in lead III. Operation was postponed for a week owing to slight coryza but was then undertaken at her urgent request.

Thoracotomy was performed on Feb. 26th and the phrenic nerve crushed. A good graft was secured and the edge of it was sutured to a well-marked and vascular sternopericardial ligament. The pericardium was opened easily, there being no tension and no adhesions. The anterior descending branch of the left coronary was much more exposed than usual and was tortuous but not calcified. The first myocardial suture was inserted to the right of this vessel without incident, although a clear view of the myocardium was obscured by subepicardial fat. A second suture was inserted and immediately bleeding was observed. The suture was at once tied, with the result that much more violent hæmorrhage occurred. It now become obvious that the wall of the right ventricle had been torn, and the bleeding point was seized with a Moynihan forceps, which tore through, leaving a large gaping wound in the ventricular wall. Three fingers of the operator's left hand were inserted into the defect so that hæmorrhage was controlled. Chordæ tendineæ were clearly felt in the ventricular cavity. One assistant then enlarged the intercostal incision, while the other opened the pericardium widely. The heart was now completely delivered from the wound, and while venous return was controlled by manual compression of the cavæ the large rent was repaired by interrupted sutures, supplemented by free muscle graft; and in addition a smaller rent on the posterior wall of the right ventricle was repaired in a similar manner. The heart was returned, the cavæ released, and at once a small tear was apparent in the wall of the right auricle. The heart was again delivered, the cavæ again controlled, and this tear repaired. The heart was replaced and was seen to fill with blood without hæmorrhage. During the whole manœuvre the total amount of blood lost was small, and the auricles were observed to be contracting again rhythmically. Cardiac massage and intracardiac adrenaline failed to restore ventricular action. The estimated duration of caval compression was 3½ and 1½ minutes.

At *post-mortem examination* the brain showed an area of old softening in the upper aspect of the posterior part of the right frontal lobe, and there was severe atheroma of the cerebral arteries. The heart weighed 10 oz., and the myocardium very friable. There were two tears in the ventricular wall, both sutured and one also plugged with a portion of voluntary muscle, and there was a subpericardial hæmatoma over the left auricle close to a sutured rent in the coronary sinus. Severe coronary atheroma was present. There was considerable atheroma of the lower abdominal aorta. The lungs were congested and showed basal collapse, especially on the left side. The kidneys and their vessels appeared normal.

CASE 8

A machine-hand, aged 56, was admitted to Lambeth Hospital from Dr. W. G. Watson on Feb. 20th, 1937, complaining of shortness of breath for eighteen months and pain over the heart, on exertion only, for six months. The pain lasted about two minutes and radiated into the left arm; it was never agonising, had never occurred at rest, and was more likely to occur after a heavy meal. There were no other symptoms. There were no previous illnesses and he was passed A1 in the war. There was no history of angina in the family.

Physical *examination* showed a well-nourished man with a high colour. The tongue was moist and clean and his teeth had all been extracted about three years before for neuralgia. The heart was not enlarged, the aortic second sound was thin and sharp, and there was a systolic murmur at the apex. Frequent extrasystoles were present. There was moderate radial and brachial arterio-sclerosis, and the blood pressure was 150–130 mm. systolic and 75 diastolic. Nothing abnormal was found in the abdomen and central nervous system. There was very slight

pitting over the left tibia. The urine and kidney function tests were normal and the blood-urea 28 mg. per 100 c.cm. The Wassermann reaction was negative. X ray examination showed a long thorax, with nothing abnormal in the heart, lungs, or aorta. The electrocardiogram showed some slurring of S in all leads and numerous premature ventricular contractions. He was diagnosed as an early case of angina pectoris.

Operation was performed on March 5th, the pericardium being opened via Larrey's angle and aleuronat being

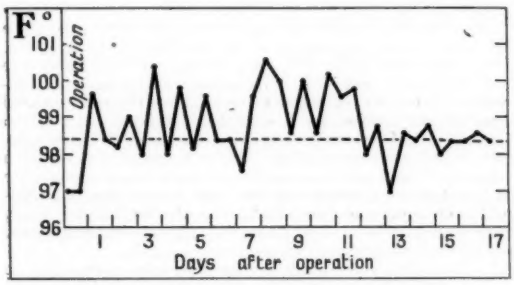

FIG. 6.—Typical post-operative temperature chart.

inserted. There was pyrexia (Fig. 6) for eleven days and the extrasystoles became more numerous. A soft pericardial rub was heard and X ray examination suggested a small pericardial effusion. He was allowed up after three weeks and discharged home on April 6th. He had had no pain since the operation but extrasystoles were still present. The electrocardiogram showed marked elevation of the S–T interval, especially in lead II, during the first week, and this was followed at the end of three weeks by flattening or inversion of the T waves in all leads. Three months after operation the T waves had returned to normal in leads I and II but remained inverted in lead III.

There has been no pain since operation. He complained of some breathlessness on exertion for the first three months but is now symptom-free and back at work.

CASE 9

A secretary, aged 50, was admitted to Lambeth Hospital on March 7th with a history of anginal attacks for four years. Two attacks had been particularly severe: one eighteen months before, when the pain had occurred at rest and lasted 48 hours and he was in bed for three weeks; and the other four weeks before admission, when the pain again came on during rest, lasted 24 hours, and he was in bed for two days. An attack was "always hanging about," and his activity was greatly curtailed. Amyl nitrite had given some relief. He had been compelled to move into a bungalow to avoid stairs and had given up driving a car. His previous history was negative, except for influenza in 1918. His father had one stroke before he died of chest trouble, and his mother died of a stroke. A maternal uncle died suddenly of a severe pain in the chest.

Physical *examination* showed an obese man, 5 ft. 4½ in. in height and weighing 12 st. 4 lb. The heart was not enlarged, the sounds were normal, and there were no murmurs. The blood pressure was 140 mm. systolic and 100 diastolic, and there was no arterio-sclerosis. Nothing abnormal was found in the chest, abdomen, and central nervous system. The urine and kidney function tests were normal and his blood-urea was 36 mg. per 100 c.cm. The Wassermann reaction was negative. X ray examination showed an enlarged heart. The electrocardiogram revealed well-marked Q deflections, with inversion of the T waves in lead III and low T waves in lead II. Lead IV was normal. The findings suggested an old coronary thrombosis of T_2 type.

Cardio-omentopexy was carried out on March 12th. Owing to a very large amount of pericardial fat the phrenic nerve was not seen, but it was crushed successfully and a large fat omental graft was obtained. There were no pericardial adhesions. Aleuronat was used, and the graft was not sutured to the heart but only to the pericardium. The blood pressure three hours after operation was 130 mm. systolic and 88 diastolic. He developed a mild purulent bronchitis. Pyrexia lasted for a fortnight, due partly to a quinsy which arose during the second week. X ray examination on the fourth day suggested a small pericardial effusion. Serial electrocardiograms showed that the T waves became inverted in leads I and II and subsequently recovered. He was allowed up at the end of four weeks and was discharged home on April 17th, his blood pressure being 155 mm. systolic and 110 diastolic.

He is now symptom-free and has had no angina since the operation. He has moved into a larger house and is driving a car again.

CASE 10

A shipping messenger, aged 41, was admitted to Lambeth Hospital from Dr. A. W. Holthusen of Westcliff on April 10th, 1937, complaining of attacks of angina since July, 1935. The pain "gripped him like a vice" and radiated down both arms. It was brought on by exertion, and had begun, with increasing frequency, to awaken him at night. He could walk no further than 100 yards and needed 48 nitroglycerin tablets a week. He had had no previous illnesses. His father had suffered from angina but had had no attacks subsequent to a severe attack of probably coronary thrombosis on May 20th, 1933.

Physical *examination* showed a big man of rather flabby musculature. His tongue was coated. The heart was not enlarged and there were no murmurs. The aortic second sound was accentuated. The radial and brachial arteries were somewhat tortuous and the blood pressure was 184 mm. systolic and 110 diastolic. Nothing abnormal was found in the chest, abdomen, and central nervous system. There was no œdema. The urine and kidney function tests were normal. The blood-urea was 44 mg. per 100 c.cm. The Wassermann reaction was negative. X ray examination showed heavy hilar shadows and an increase of the heart shadow to the right. The electrocardiogram was of large amplitude and showed notched Q deflections in lead III. The T waves were low in lead I, flat in lead II, and diphasic in lead III, and there was depression of the S–T interval in leads I and II.

Cardio-omentopexy was performed on April 16th. There were some pleural adhesions. The heart was large, the pericardium tense but free, and there was a well-marked inferior sternopericardial ligament. A considerable amount of aleuronat was inserted, and the graft was sutured to the pericardium but not to the heart. The blood pressure at the end of operation was 112 mm. systolic and 70 diastolic. During the night the pulse-rate rose to 144 and the systolic blood pressure the next morning was only 68. Febrile reaction lasted over a week and leucocytosis reached 25,600 per c.mm. A soft pericardial rub was heard on the third day. On the sixth day his general condition and the physical signs suggested a left hæmothorax, and this was confirmed by paracentesis and X ray examination. Convalescence was slow. He was allowed up at the end of six weeks and was discharged home on May 29th. The electrocardiogram during the first week showed marked depression of the S–T interval in lead I and elevation in lead III.

Fourteen weeks after operation he looked well but was getting mild anginal attacks both on exertion and at rest. The heart sounds were tic-tac and a definite gallop rhythm was present. The blood pressure was 168 mm. systolic and 100 diastolic. A month later the gallop rhythm had disappeared but the heart sounds were still tic-tac. He returned to light work on Sept. 30th, five and a half months after operation. He is now getting no serious pain and can climb the 72 stairs to his office without pain or distress. The heart sounds are of good quality and the blood pressure 155 mm. systolic and 105 diastolic. The left diaphragm still shows paradoxical movement.

CASE 11

A tool-maker, aged 56, was admitted to Lambeth Hospital from Dr. F. B. Mallinson on Feb. 20th, 1937, with a history of angina pectoris for four years. Attacks were becoming more frequent and might stop him three or

four times during his twenty minutes' walk to the station. He had had pleurisy twenty years ago and renal colic three years later, when he passed three small stones. He had been a non-smoker since the age of 19 and was a total abstainer. There was no family history of angina.

On *examination* he appeared a well-nourished, grey-haired man. The apex-beat was in the 5th intercostal space, 4½ in. out, and the cardiac impulse was heaving. The aortic second sound was thin and there was a soft systolic murmur at the apex. The arteries were not thickened and the blood pressure was 140 mm. systolic and 70 diastolic. Nothing abnormal was found in the chest and abdomen. The ankle-jerks were not obtained. There was no œdema, the urine and kidney function tests were normal, and the blood-urea 32 mg. per 100 c.cm. The Wassermann reaction was negative. X ray examination showed very heavy hilar shadows and moderate enlargement of the heart. The electrocardiogram showed left axis-deviation, with notching of the S deflections in lead III. The T waves were inverted in lead I (in June, 1935, they were known to have been upright).

He was discharged home for domestic reasons on Feb. 28th and was readmitted on April 20th. He had been working hard without severe pain, but the exertion of the journey (carrying a light suitcase half a mile) brought him to a standstill with quite a severe attack. The electrocardiogram showed small R and notched S deflections in lead IV.

Cardio-pneumonopexy was carried out on April 23rd, omentopexy being prevented by numerous fine adhesions between lung and mediastinum. The phrenic nerve was crushed. The pericardium was not tense, the heart free, slightly enlarged, and yellowish in colour. The lower part of the left upper lobe and the upper part of the lower lobe were sutured to the cut edge of the pericardium, after insertion of aleuronat. The blood pressure at the end of operation was 125 mm. systolic and 75 diastolic. Recovery was uneventful, with moderate pyrexia and leucocytosis. He was allowed up at the end of four weeks and was discharged home on June 9th, having had no further attacks of angina. His blood pressure was 144 mm. systolic and 84 diastolic. The electrocardiogram had shown increasing inversion of the T waves in leads I and II.

He improved at first at home and only had one attack during June, but by the end of August he was complaining of attacks as frequent as before operation and exactly similar, except that instead of stopping he could get rid of the pain by increasing his pace. He was readmitted and spontaneous pneumothorax was excluded by X ray examination. The electrocardiogram showed considerable increase of voltage, with recovery of the T waves except in lead I. The S deflections in lead IV still showed notching. He now reports that he is hard at work, but still unable to walk far without anginal pain.

CASE 13

A printer, aged 43, was admitted to Lambeth Hospital from Dr. G. A. Shepherd on April 13th, 1937, with the story that he had collapsed unconscious in his office on Jan. 21st. For a week previously he had complained of pain behind the breast-bone and two days before he had been awakened by an attack of palpitation which lasted four hours and frightened him considerably. He was treated in bed for a week and then sent to the seaside, where he complained of pain in the chest on exertion. Nearly a month after the attack he was seen by a cardiologist, who diagnosed it as coronary thrombosis and advised four weeks in a nursing-home at complete rest, followed by four weeks' restful holiday. During this period his weight was reduced from 15 st. 6 lb. to 13 st. 5 lb., and he complained of occasional attacks of pain. His previous health had always been good and there was no history of angina in the family.

On *examination* he was well nourished and appeared apprehensive and highly strung. The heart was not enlarged, the sounds were normal, and there were no murmurs. The arteries were not thickened and the blood pressure was 144 mm. systolic and 90 diastolic (compared with 130 mm. systolic and 90 diastolic in February, 1937). The chest moved rather poorly but was otherwise normal, and nothing abnormal was found in the abdomen. The tendon reflexes were exaggerated. The urine and kidney function tests were normal and the blood-urea was 24 mg. per 100 c.cm. The Wassermann reaction was negative. X ray examination showed very slight enlargement of the heart. The electrocardiogram showed well-marked Q deflections, with inversion of the T waves in lead III and low T waves in lead II. The S–T interval was rising in lead I and raised and falling in lead III. Lead IV was normal. The findings were compatible with a healing cardiac infarction of T_3 type. He was treated for a fortnight before operation with a balanced diet, breathing exercises, and a sedative mixture.

Cardio-omentopexy was performed on May 7th. The phrenic nerve was crushed and a good fatty graft obtained. The heart was large, the pericardium not tense and free of adhesions, and the myocardium fatty and greyish-yellow in colour. Aleuronat was used and the graft sutured to the pericardium only. The blood pressure at the end of operation was 110 mm. systolic and 68 diastolic. Recovery was uneventful. Pyrexia lasted for fourteen days and leucocytosis reached 21,200 per c.mm. He was allowed up at the end of four weeks and was discharged home on June 19th, the blood pressure being 120 mm. systolic and 88 diastolic. The electrocardiogram underwent no material change.

For the first three months after discharge he complained of some breathlessness on exertion, and had four attacks of mild retrosternal pain, and on examination his heart sounds were tic-tac. He is now free of symptoms and back at work, but still taking life rather more easily than he need. His heart sounds are normal and the blood pressure 130 mm. systolic and 94 diastolic. The left diaphragm has recovered its tone and movement. In the electrocardiogram Q waves and inversion of the T waves are still present in lead III, but the S–T interval is now normal in leads I and III.

CASE 14

A french-polisher, aged 56, was admitted to the London Jewish Hospital on April 9th, 1937, with a three months' history of angina pectoris. The first attack had occurred on walking, and at the time of admission they were coming on every three or four hours. The pain was very severe but was relieved by three nitroglycerin tablets. In hospital the attacks became fewer and milder. He was transferred to Lambeth Hospital on May 14th. He gave a history of influenzal pneumonia in 1918, broncho-pneumonia six or seven years ago, and winter bronchitis for many years. There was no history of angina or other cardiovascular disease in his family.

Physical *examination* showed a well-nourished, freckled man, 5 ft. 5 in. in height. He was confined to bed but was able to wash himself. Attacks were far less frequent and pain was now relieved by half a tablet of nitroglycerin. The apex-beat was in the 5th intercostal space, 4 in. from the midline. The mitral first sound was accentuated and the aortic second sound thin. There were no murmurs. Auricular fibrillation was present, the ventricular rate being 100 per min. There was no arterio-sclerosis and the blood pressure was 130 mm. systolic and 80 diastolic. The chest moved poorly but the lungs were normal. The abdomen was soft and rotund and the central nervous system normal. There was no œdema. The urine contained a trace of albumin. The blood-urea was 28 mg. per 100 c.cm. and the kidney function tests were satisfactory. The blood showed polycythæmia and gave a negative Wassermann reaction. X ray examination showed a large triangular heart shadow. The electrocardiogram showed auricular fibrillation, left axis-deviation, and notching of the Q R S complexes. The main deflection in lead IV was negative and notched. He was treated with Luminal and a venesection of one pint.

Thoracotomy was performed on May 28th. The pulse-rate rose to 160 as soon as the chest was opened, so aleuronat was inserted into the pericardium and the operation terminated. The pericardium was not tense. The heart was greyish-yellow in colour and free of adhesions.

Recovery from the actual operation was fairly uneventful. He was afebrile for a week, and then ran a mild irregular temperature for the next month, probably due to a small

pleural effusion which developed at the left base. Leucocytosis reached 14,400 per c.mm. on the third day. On the ninth day his mental state became slightly confused and never quite regained normality afterwards. By July 5th, five and a half weeks after operation, he was sitting up in the evenings and able to take a few steps round the room. He had had no further attacks of angina. Five days later he became suddenly blue and dyspnœic and mentally more depressed. Severe pleuritic pain developed on the right side, the sputum became blood-stained, and signs of pulmonary infarction were found at the right base. His condition rapidly deteriorated, thrombosis of the right femoral vein occurred on July 19th, and he died the following day.

There was no post-mortem examination.

CASE 15

A widow, aged 65, was transferred to Lambeth Hospital on April 9th, 1937, from Lewisham Hospital (L.C.C.), where she had been admitted on July 28th, 1936, for a coronary thrombosis of T_3 type occurring that day. Before the attack she had complained of breathlessness on exertion for six years and for five years attacks of precordial pain brought on by exertion as trivial as sewing. Since the occlusion she had remained confined to bed and had complained of a persistent feeling of constriction round the heart, with exacerbations of pain needing morphia. She gave a history of several pelvic operations, and Mr. Cecil Joll had removed a goitre 20 years ago. She had had winter bronchitis of late years and was subject to laryngitis. Her father had dropped dead of heart trouble.

Physical *examination* showed an obese woman, confined to bed and becoming easily dyspnœic on slight exertion. The heart showed accentuation of the first sound and a systolic murmur audible at the apex and in the aortic area. The blood pressure was 154 mm. systolic and 90 diastolic. There were râles at the right base. The abdomen was normal. She complained of paræsthesiæ in the arms, and there was diminished sensibility to pinprick and cotton-wool. The urine and kidney function tests were normal and the blood-urea was 28 mg. per 100 c.cm. The Wassermann reaction was negative. X ray examination showed enlargement of the cardiac shadow to the right, ? aneurysmal. The electrocardiogram showed left axis-deviation. The T waves were still inverted in lead III but the S-T interval was now normal.

A month after admission she had an attack of severe constricting pain round the heart and lost consciousness for about two minutes. Her pulse and colour were very poor at the time but she recovered well. Attempts were made to activate her but even the exertion of getting out of bed brought on an attack of pain.

Cardio-omentopexy was performed on June 4th. The phrenic nerve was crushed and a fatty omental graft easily obtained. The inferior sternopericardial ligament was well marked. The pericardium was fatty and rather tense over a slightly enlarged heart. The heart was free and showed no evidence of infarction. The graft was sutured to the heart without aleuronat. The pulse-rate at the end of operation was 80 and the blood pressure 105 mm. systolic and 68 diastolic. She developed a painful non-productive cough with much bronchial spasm, and dyspnœa and cyanosis on movement, but no signs of consolidation were found. The blood pressure had returned to its pre-operative level within a week. Dyspnœa on exertion persisted for about a month. The electrocardiogram showed flattening of the T waves in leads I and II on the tenth day but recovery to normal in three weeks. Activation was begun seven weeks after operation and she sat out of bed at the end of two months for the first time for over a year. She still complained of an occasional feeling of heaviness across the chest but no anginal attacks. She was transferred to St. Benedict's Hospital for convalescence on Oct. 22nd, having lost 1 st. by dieting.

She now reports that she is up and about the ward three or four hours a day without symptoms.

CASE 17

A traveller, aged 65, was admitted to Lambeth Hospital on June 29th, 1937, with a history of angina pectoris for a year. Attacks had become so frequent, even occurring at rest, that in January, 1937, he had been admitted to hospital for treatment by paravertebral alcohol nerve-block. He had had no pain since, but was now complaining of increasing breathlessness on exertion and was unable even to shave himself. There was no history of previous illness and no family history of angina.

On *examination* he appeared a florid, grey-haired man. The heart was not enlarged clinically, the sounds were rather toneless, and there were no murmurs. Moderate arterio-sclerosis was present and the blood pressure was 132 mm. systolic and 88 diastolic. There were râles at both bases. The vital capacity was 1900 c.cm. and the chest expansion 1 in. The liver was just palpable but not tender. The knee-jerks were not obtained. The urine contained a trace of albumin. The blood-urea was 40 mg. per 100 c.cm. and the urea-concentration test 2·1 per cent. and 2·6 per cent. The Wassermann reaction was negative. X ray examination showed enlargement of the heart. The electrocardiogram showed left axis-deviation and notching of the Q R S complexes. The T waves were inverted in lead I and low in lead II. The main deflection in lead IV was negative.

Cardio-omentopexy was performed on July 2nd. The phrenic nerve was crushed but the diaphragm did not rise so high or so dramatically as usual. The pericardium was tense but free of adhesions. Aleuronat only was used for application of the graft, and the inferior sterno-pericardial ligament and lung fell nicely into apposition. The latter was unusually large, voluminous, and tough. The pulse-rate at the end of operation was 96 and the blood pressure 72 mm. systolic and 52 diastolic.

His condition was satisfactory until the fourth day, when signs of consolidation of the left lower lobe developed. On the seventh day a slight purulent discharge began from the sternal end of the wound; this proved ultimately to be due to necrosis of the 6th costal cartilage. The pneumonia ran a satisfactory course until July 20th, when he became lethargic and on examination showed a dry tongue, pulse less good, consolidated left lower lobe, enlarged and tender liver, slight œdema of the ankles and lumbar region, and much albuminuria. The blood pressure was 114 mm. systolic and 78 diastolic. The blood-urea was 68 mg. per 100 c.cm. In four days all œdema had disappeared but he remained drowsy. Two days later an empyema burst through the outer end of his wound and his mental condition improved at once. A hæmolytic streptococcus was isolated from the pus and 5 c.cm. of Prontosil Soluble was injected on two occasions into the empyema cavity. He seemed to be improving slowly, but necrosis of the costal cartilage was progressive and on August 22nd his pulse was noted *for the first time* as being less good. He died suddenly in his sleep three days later, 7½ weeks after operation.

At *post-mortem examination* the operation wound was found to have healed except for two large sinuses; one at the sternal end exposed the 6th costal cartilage, which was ununited to its rib and in a condition of necrosis; the other led backwards into the axilla to a sinus entering the pleural cavity. The heart was rather small and the myocardium soft and fibrotic. Both coronaries were calcified for the first 2 in. of their course, and their lumina were almost completely obliterated. There was one small superficial adhesion high up over the left ventricle and a dense tubular adhesion to the anterior aspect of the apex. This corresponded outside the pericardium to a densely adherent mass consisting of fatty inferior sterno-pericardial ligament, portion of left lung, left dome of diaphragm, and thoracic end of the graft. There was a small empyema cavity, empty of pus, over the lower half of the left lower lobe. The upper lobe of the left lung was normal. Its lower lobe was collapsed and the cut surface showed thickened and dilated bronchioles protruding from the retracted lung parenchyma. The right lung showed basal congestion and œdema. The aorta showed diffuse early atheroma. In the abdomen the omental graft passed through the left leaf of the diaphragm near its lateral attachment. The liver was fatty and the spleen toxic and diffluent. The kidneys were rather large and showed congestion on section. The capsule stripped easily, leaving a smooth surface. On histological examination the myocardium showed extensive fibrosis and areas of

recent infarction. There were many patent blood-vessels in the graft lying over the epicardium, and there was also a very vascular subepicardial zone corresponding to it, as seen in Case 2. The specimen is receiving further histological study.

CASE 18

A housewife, aged 62, was admitted to Lambeth Hospital on April 23rd, 1937, with a six years' history of angina pectoris. During the last year her activity had been so much curtailed that she had been unable to go shopping. Pain had occasionally occurred in bed. She was also very short of breath, had a bad cough, and slept badly. She was in bed for three months with rheumatic fever at the age of 18. There was no family history of angina. She continued to have attacks of angina while in hospital, and after a severe attack on June 30th she was referred to the clinic.

Physical *examination* showed a rather obese and florid woman. There was gross oral sepsis. The heart was enlarged, the sounds were soft and spaced with a well-marked gallop rhythm, and there was a very soft mitral systolic murmur. The arteries were not thickened and the blood pressure was 186 mm. systolic and 106 diastolic. There were râles at both bases; the vital capacity was 1500 c.cm. and the chest expansion ¼ in. The abdomen was normal and the central nervous system also except for small irregular sluggish pupils. There was no œdema. The urine and kidney function tests were normal and the blood-urea 26 mg. per 100 c.cm. The Wassermann reaction was negative. X ray examination showed an enlarged heart. The electrocardiogram showed left axis-deviation and inversion of the T waves in lead I. The duration of Q R S was 0·12 sec.

All her teeth were extracted. When re-examined before operation, gallop rhythm was only just discernible, but the heart sounds were still tic-tac. There were very few basal râles and the blood pressure was 164 mm. systolic and 90 diastolic.

Cardio-omentopexy was performed on July 2nd. The phrenic nerve was crushed and a good graft obtained. The lung was emphysematous and a bulla could be seen posteriorly. The pericardium was tense and contained excess of fluid. The surface of the heart showed a pale area. Aleuronat was applied and the graft sutured to the edge of the pericardium. A well-marked inferior sterno-pericardial ligament and the lung fell nicely into apposition. The pulse-rate at the end of operation was 110 and the blood pressure 104 mm. systolic and 66 diastolic.

Her immediate progress was good. She developed purulent bronchitis and a urinary infection, both of which settled down. Tachycardia and leucocytosis however, persisted for no obvious cause, and three and a half weeks after operation she had an anginal attack lasting fifteen minutes. Her condition then slowly improved and by August 9th she had been up twice without ill effects. The following day she had two further attacks and was confined to bed again. Eight weeks after operation she was still confined to bed and was getting frequent attacks of pain, relieved by nitroglycerin. On examination the heart was not enlarged. There was tachycardia but no gallop rhythm. Very slight pitting œdema was present over the tibiæ. The blood pressure was 150 mm. systolic and 84 diastolic. She died suddenly on Sept. 6th, two months after operation.

At *post-mortem examination* the wound was found soundly healed. The left lung and the inferior sternopericardial ligament were adherent to the graft, which was itself adherent to the heart. There was complete adhesion of the pericardium, and on separation of part of it several bleeding points could be seen on the surface of the heart. The heart showed hypertrophy of the left ventricle, with diffuse fibrotic changes in the myocardium. The coronary tree showed diffuse sclerosis and atheroma, but detailed examination of its ramifications was not carried out as the specimen is being preserved for further histological study of the adhesions, which initial examination shows to be highly vascular. The lungs were normal. The abdominal viscera were not abnormal and the graft could be seen passing through the diaphragm at its normal site. The colon was not kinked.

CASE 19

An engineer, aged 55, was admitted to Lambeth Hospital from Dr. Blair Macaulay of Liverpool on July 26th, 1937, complaining of breathlessness on exertion of gradual onset, 3½ years ago, when working long hours under bad conditions on the Mersey Tunnel. Soon afterwards he had begun to get attacks of pain across the chest on exertion. At the time of admission he could not walk more than twenty or thirty yards without inducing an attack. His previous history was negative except for a herniotomy ten years ago, and there was no family history of angina.

Physical *examination* showed a lean but well-nourished man of florid complexion. The tongue was moist and clean, but the gums round his six remaining teeth showed pyorrhœa. The heart was not enlarged, the sounds were only just audible, and there were no murmurs. The radial and brachial arteries were moderately thickened and the blood pressure was 100 mm. systolic and 70 diastolic. There were a few persistent râles at the right base and a constant cough was noticeable. The chest expansion was 1½ in. Nothing abnormal was found in the abdomen. The urine contained a trace of albumin. The blood-urea was 44 mg. per 100 c.cm. and the urea-concentration test was normal. The Wassermann reaction was negative. X ray examination showed a normal heart shadow. The electrocardiogram showed a low voltage, the Q R S complexes measuring 1 mm. in lead I. The T waves were upright in all leads but low in lead I.

He was treated with breathing exercises and a stimulating expectorant mixture, and his remaining teeth were extracted. His cough and sputum persisted, however, and it was decided that he was only fit for pericardiotomy via Larrey's angle. This operation was advised in the hope that he might improve sufficiently to allow cardio-omentopexy to be performed at a later date. His vital capacity before operation was 3150 c.cm.

Pericardiotomy via Larrey's angle was performed on August 28th. The heart was greyish-yellow in colour. In spite of the minor nature of the operation he developed purulent bronchitis with mild pyrexia but no leucocytosis. He was allowed up for the first time two and a half weeks after operation and was discharged home for personal reasons on Sept 23rd, his blood pressure being only 96 mm. systolic and 66 diastolic. He was able to walk round the grounds and up one flight of stairs without pain or breathlessness.

His improvement was short-lived and he now reports that his condition is as bad as before the operation.

To sum up, of these fifteen cases of angina submitted to operation we record five deaths: one of uræmia in a man aged 72, three months after operation; the second at operation through rupture of a friable ventricle; the third of pulmonary infarction two months afterwards; the fourth suddenly in the second month, following a stormy convalescence complicated by pneumonia, empyema, and necrosis of a costal cartilage; and the last suddenly for no apparent cause, again two months after operation. On the other hand, of the ten who lived we have knowledge that eight have eventually enjoyed freedom from angina, and of the eight men seven are back at work.

(To be concluded)

MENTAL AFTER-CARE ASSOCIATION.—The object of this association is to provide some form of assistance, including homes for convalescence, for the poor treated in public mental hospitals to bridge over the difficult period intervening between recovery and return to normal life. Later some are helped to find suitable employment. This year nearly 4000 people have been befriended by the association. It also assists authorities who are adopting the boarding-out system for chronic patients. An earnest appeal for funds to carry on and extend this work is being made. Contributions should be sent to Miss E. D. Vickers, 354, Grand Buildings, London, W.C.2.

A NEW SYNDROME APPARENTLY DUE TO OVER-ACTIVITY OF THE POSTERIOR PITUITARY

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A PATIENT presenting the combination of hypertension, hyperchromic anæmia, achlorhydria, and abnormal carbohydrate tolerance has been investigated for the past eight months and, as this syndrome has not been previously reported, a preliminary account is given here.

CASE-HISTORY

A. B., aged 26, an insurance broker, was first seen on March 7th, 1937, complaining of "not feeling fit" and of nose-bleeding.

He had had intermittent epistaxis for a week in March, 1937, and since then had had lethargy, discomfort in the lumbar region, and occasional diffuse headaches. There was undue dyspnœa on exertion and he had a slight cough. There were no urinary or gastro-intestinal symptoms but he had occasional "thumpings of the heart." He smoked 15–20 cigarettes daily. His weight was stationary and he had a good appetite with a preference for fatty foods and an avoidance of salt at all times. The bowels acted regularly. Previously he had always been well; he could not date the definite onset of the symptoms apart from the epistaxis. His father died of "angina" at 55; his mother and one brother were alive and well.

TABLE I—SUMMARY OF BLOOD COUNTS

Date.	Hæmoglobin (%).	Erythrocytes (per c.mm.).	Colour-index.	Leucocytes (per c.mm.).
March 7th .. (Liver given)	78	3,400,000	1·26	2800
March 15th ..	86	4,080,000	1·16	9100
„ 23rd ..	84	4,770,000	1·04	6300
„ 30th .. (Liver discont.)	92	5,430,000	0·93	3500
April 5th .. (Liver given)	88	3,320,000	1·45	3850
April 15th ..	102	4,650,000	1·18	5250
May 11th ..	92	5,480,000	0·92	7700
„ 30th ..	100	4,840,000	1·14	4900
July 25th ..	80	5,500,000	0·80	4900
August 7th ..	100	5,040,000	1·08	—
Sept. 22nd ..	100	5,030,000	1·08	6650

discont. = discontinued.

Examination.—The patient is a lean athletic man of sallow complexion with hair thinning in the frontal region and the vertex. Height, 5 ft. 10 in.; weight, 155 lb.; tongue normal; teeth good. Pulse 72, regular and of good tension, arteries not obviously thickened; apex-beat 4 in. from the midline in the fifth intercostal space; no cardiac enlargement to the right of the sternum; heart sounds normal apart from accentuation of the aortic second sound; blood pressure in both arms 190/110, and in the legs 200/110. Lungs: nothing abnormal discovered. Abdomen: ? tip of spleen just palpable. Nervous system: reflexes all present and brisk; sensation normal; pupils dilated, reacting briskly to light and accommodation. Fundi: veins full, arteries somewhat narrowed; no hæmorrhages or exudate. Urine: sp.g. 1022, *p*H 5·7; no albumin, sugar, acetone, or blood; trace of urobilinogen; oxalate crystals ++; chlorides ++.

SPECIAL INVESTIGATIONS

Radiography showed clear lung fields and a heart-lung ratio within normal limits; the pituitary fossa was normal, almost roofed over, and the sinuses were clear.

Capillary resistance (tourniquet test): strongly positive.

Blood counts are summarised in Table I.

Gastric residuum (six hours after last meal).—May 6th, 1937: 8 c.cm. of opalescent fluid removed with difficulty; no free HCl; total acids (combined acid) equivalent to 1·5 c.cm. of N/10 NaOH or 0·0054 per cent. HCl. After 0·25 mg. histamine subcutaneously only 5 c.cm. of gastric juice could be obtained twenty minutes later; no free HCl; total acids equivalent to 2·3 c.cm. of N/10 NaOH or 0·0083 per cent. HCl. The two samples were similar in appearance and there was a considerable quantity of mucus.

TABLE II—BLOOD-SUGAR CURVE OF THE PATIENT IN MAY, 1937

Fasting level.	Number of minutes after 50 g. of glucose were given.			
	30	*60*	*90*	*105*
129 mg.	338 ..	286 ..	314 ..	270

TABLE III—BLOOD-SUGAR CURVE OF THE PATIENT IN SEPTEMBER, 1937

Fasting level.	Number of minutes after 50 g. of glucose were given.			
	30	*60*	*90*	*120*
146 mg.	238 ..	177 ..	228 ..	204

Fæces.—Formed, brown, no abnormal amount of mucus, no blood; large numbers of soap flakes present and a few fat droplets. Fat estimation *on dried fæces* (May 23rd): total fat 31 per cent., total fatty acids 21 per cent., and neutral (unsplit) fats 10 per cent.

Renal function.—Early morning fasting specimen: sp.g. 1022, volume 150 c.cm., urea 3 g. per 100 c.cm. Then 15 g. of urea were taken in 100 c.cm. of water, and 1 hr. 50 min. later sp.g. was 1020, volume 150 c.cm., and urea 3 g. per 100 c.cm.; 3 hr. 30 min. later sp.g. was 1022, volume 113 c.cm., and urea 2·85 g. per 100 c.cm. (The patient found it impossible to pass urine at hourly intervals after the dose of urea.) Water-elimination test: after ingestion of 20 oz. water, 16·5 oz. of urine was passed within three hours. Blood-urea: 86 mg. per 100 c.cm.

Blood-sugar curves.—See Tables II and III. (Method of Hagedorn and Jensen on capillary blood.)

Further tests on August 8th.—Van den Bergh: direct reaction negative. Blood chlorides: 612 mg. per 100 c.cm. (Van Slyke method). Serum calcium: 8·8 mg. per 100 c.cm. (Clark and Collip).

COMMENT AND DISCUSSION

The combination of a hyperchromic anæmia and hypertension in a patient aged 26 was at first thought fortuitous; but the fact that Dodds, Noble, and others (1935a and b) had produced a hyperchromic anæmia in animals by means of posterior pituitary extract made one consider the possibility of hyperfunction of the posterior pituitary. Cutting, Dodds, and others in 1937 also produced achlorhydria with the extract, and the achlorhydria of this patient, together with his abnormal carbohydrate tolerance, strengthened this possibility, on which the following discussion is based.

The blood picture (Table I) was at first that of a hyperchromic anæmia. After administration of liver the blood count returned rapidly to normal, but on discontinuing liver the red cell count fell again. It was restored to normal when liver was again given, and though liver therapy was entirely discontinued after a month the blood picture has remained approximately normal since. Associated with this anæmia was a histamine-refractory achlorhydria. The van den Bergh reaction was unfortunately not done at the time when the patient was anæmic.

Hypertension.—The blood pressure in April, 1937, was 190/110; it fell to 164/110 in July and returned to normal in September (Table IV). It will be seen

TABLE IV—BLOOD-PRESSURE READINGS

April 8th ..	190/110	July 1st ..	164/110
„ 23rd ..	205/110	„ 13th ..	165/100
May 13th ..	165/108	August 16th ..	160/104
June 1st ..	162/105	Sept. 23rd ..	130/80

from a comparison of Tables I and IV that in April, when the blood pressure was high, the blood picture was hyperchromic, and in September, when the pressure had returned to normal, the blood picture was normal.

Carbohydrate tolerance.—Tables II and III are representative blood-sugar curves. When the patient was first seen (Table II) his carbohydrate tolerance was grossly abnormal, but later (Table IV) when the blood pressure had fallen, the curve (Table III) is not nearly so abnormal. The patient has exhibited no symptoms of diabetes and his weight has remained constant for the last three years.

Carbohydrate metabolism and the pituitary gland.—The association of the anterior lobe of the pituitary with carbohydrate metabolism is well known, Houssay (1936) and others having shown that injections of anterior lobe extract produce a "diabetic" condition in normal animals, and Young (1937) having actually produced permanent diabetes in dogs after the injections were stopped. The relation of the posterior lobe extract (Pituitrin) to carbohydrate metabolism is less clear and somewhat disputed. Burn (1923), working on rabbits, demonstrated the antagonism of pituitrin to insulin, and Lawrence and Hewlett (1925), working on normals and diabetics, demonstrated the same antagonistic effect but found no appreciable or constant effect on the blood-sugar when pituitrin was injected without insulin. Lambie (1926), however, showed that pituitrin does elevate the blood-sugar and this in turn has been disputed.

Diabetes and anæmia.—A hyperchromic anæmia with diabetes is rare and is usually regarded as fortuitous. Wilkinson (1933) found that about 1 per cent. of patients with pernicious anæmia had diabetes, and recent correspondence in the *British Medical Journal* confirms the rarity of this association.

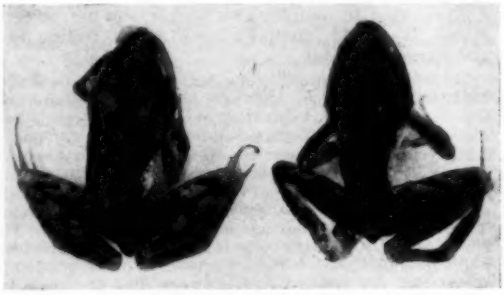
Anæmia and hypertension.—The association of a hypertension with a hyperchromic anæmia is also rare. Wilkinson found only 8 cases of hypertension among 370 cases of pernicious anæmia. Reinhart (1934–35) described pernicious anæmia in 3 elderly patients who had hypertension; their urine was normal and no tests were made for pressor substances. Dodds and others (1935 a and b), and Cutting, Dodds, and others (1937), have shown that injection of a posterior pituitary extract produces a hyperchromic anæmia in rabbits and also inhibition of the secretions of the stomach and pancreas. The vasopressor and antidiuretic effects of this extract are well known and in addition it also causes dispersion of the melanosomes in frogs.

PITUITRIN-LIKE ACTION OF AN EXTRACT OF THE PATIENT'S URINE

1. *Pressor action.*—In view of the findings in this patient, Dr. Noble was asked to test the blood and urine of the patient for pressor principle. This he kindly consented to do and his methods and results are given in detail in the accompanying paper. From the urine he extracted a pressor principle which caused a persistent elevation of the blood pressure of the cat, the tracing obtained being essentially similar to that obtained with posterior pituitary extract.

2. *Antidiuretic action.*—The extract also had a pronounced antidiuretic action on the rat, and when compared with the action of pituitrin it was found that the pressor and antidiuretic principles appeared to vary independently of each other.

3. *Melanosome-dispersing action.*—As the extract gave two of the actions of pituitrin its effect on the melanophores of the frog was investigated. With the coöperation of Prof. St. G. Huggett, I injected two frogs (*Rana temporaria*) with 0·1 and 0·5 c.cm. of the extract of the patient's urine. The control frogs remained a normal colour while the other frogs



Frog A was injected with 0·1 c.cm. of an extract of normal urine, while frog B was injected with 0·1 c.cm. of an extract of the patient's urine. Note the dark colour of frog B which is due to the dispersal of the melanosomes.

became very dark (see Figure) indicating the presence in the extract of a melanosome-dispersing principle.

4. *Other properties of the extract.*—Dr. Noble found that the pressor action of the extract was unaffected by boiling. This is also true for posterior pituitary extract. It should be noted that in September, when the hypertension had disappeared, the blood picture had returned to normal and the carbohydrate tolerance was better, the amount of pressor and antidiuretic substances in the urine had also very noticeably decreased.

PREVIOUS WORK ON PRESSOR SUBSTANCES IN THE BLOOD AND URINE

The question of pressor substances in the blood and urine has engaged attention for some years. Bohn (1931–32) and Coester (1934) claim to have detected pressor substances in the blood of "pale hypertensives" which were absent in the blood of normal persons and "red hypertensives." This work was not confirmed by de Wesselow and Griffiths (1934) who found, using Bohn's method, that a pressor principle can be detected in the blood of normal individuals, chronic glomerulo-nephritics, and malignant and essential hypertensives. Capps and others (1934) have also reinvestigated Bohn's findings and have not confirmed his results. The curves they obtained were very variable and they could find no correlation between them and the curves obtained with adrenaline, pituitrin, saline, or the carotid sinus reflex.

CONCLUSIONS

An extract of the patient's urine contained a substance or substances which had the actions of posterior pituitary extract and the clinical findings of hyperchromic anæmia, hypertension, and abnormal carbohydrate tolerance suggest that there is hyper-

function of the posterior lobe of the pituitary. The fact that as his condition improved the amount of pituitrin-like substance excreted in his urine diminished indicates that the hyperfunction is either an isolated occurrence or is possibly cyclical in nature. Further observation of this patient over a long period will settle this point.

The syndrome described is thought to be new to clinical medicine, but the diseases that are known to affect the anterior lobe of the pituitary and other ductless glands lead one to believe that the hyperfunction of the posterior pituitary is at least theoretically possible. Some writers have affirmed that the posterior pituitary has no action in the normal body, but the work of Gilman and Goodman (1937) in demonstrating the presence of an antidiuretic substance in the urine of dehydrated rats, whereas hypophysectomised dehydrated rats have none, shows that it has a very definite action.

SUMMARY

A patient is described in whom hypertension, hyperchromic anæmia, achlorhydria, and abnormal carbohydrate tolerance is associated with the excretion in the urine of a substance which has the actions of posterior pituitary extract.

It is suggested that the syndrome is due to hyperfunction of the posterior pituitary.

I am deeply indebted to Miss Nora G. Sproston who first detected the anæmia and who has kindly carried out all the pathological investigations described.

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CLINICAL HYPERFUNCTION OF THE POSTERIOR LOBE OF THE PITUITARY

SUGGESTED BY A PRESSOR AND ANTIDIURETIC SUBSTANCE OBTAINED FROM THE URINE

BY R. L. NOBLE, M.D. Toronto, Ph.D. Lond.*

H. RINDERKNECHT

AND

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IN the preceding paper Dr. E. I. Jones has described clinical findings which it is suggested are due to a hyperfunction of the posterior lobe of the pituitary gland. The case he reports presented the following features: hypertension, hyperchromic macrocytic anæmia which responded to liver therapy, achlorhydria, and abnormal carbohydrate tolerance. Experimentally the pressor effect of posterior pituitary extracts is well recognised; similarly a hyperglycæmic action has been demonstrated. The production of achlorhydria following injections of the vasopressive principle of posterior lobe extracts (Dodds and Noble 1937; Cutting, Dodds, Noble, and Williams 1937) which may in some animals be followed by a severe hyperchromic macrocytic anæmia (Dodds and Noble 1935) has been recorded. The similarity between the condition of this patient and that which could be produced in animals suggested that it would be of interest to determine whether the vasopressive principle could be obtained from the patient's blood or urine.

The recovery of antidiuretic substances from the blood of patients with toxæmia of pregnancy by ultrafiltration has been reported by some workers (Anselmino and Hoffmann 1931) and denied by others (Byrom and Wilson 1934). It is obvious from these and other papers that the recovery of active material was extremely variable using methods of ultrafiltration. Lately Melville (1937) has used 80 per cent. ethyl alcohol to extract the activity from heparinised blood, with apparently good results. The blood from this patient was therefore extracted according to his description. The untreated serum was also tested.

The urine has been extracted by a method similar to that described by Kamm (1928) and his associates for obtaining the different pituitary principles from posterior lobes.

After concentration of the acidified urine, the activity is precipitated with ammonium sulphate. This precipitate is dried and the active substance extracted with glacial acetic acid. The activity is precipitated from this by ether and petroleum ether. This dried precipitate is then extracted with 0·25 per cent. acetic acid, which contains the active substance.

This method was used on the earlier specimens of urine and was therefore used throughout, although it was later found in control experiments that the yield of active substance by this technique is under 50 per cent.

The antidiuretic activity has been assayed on rats by the method described by Burn (1931). Pressor activity has been measured on the spinal cat, using Pitressin as a standard. Samples of blood have been tested on two occasions—the first at the end of May when clinically the findings were most abnormal, and the second at the end of July when considerable clinical improvement had occurred.

Blood.—The May specimen of unextracted blood-serum was not tested for antidiuretic activity, but the intravenous injection of 5 c.cm. of serum produced a depressor effect followed by a rise of 20 mm. Hg in a spinal cat. This rise in pressure was not maintained, as is found in a typical pituitary effect, so the results were only suggestive of a pressor substance in the blood.

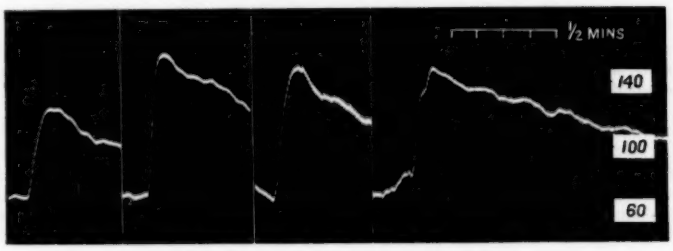
The June specimen of blood-serum was tested for antidiuretic activity and a dose of 0·25 c.cm. gave no effect. The serum similarly gave no pressor effect after the injection of 1 c.cm. Part of this specimen was extracted by Melville's method. It was found that the equivalent of 4 c.cm. of blood gave no antidiuretic effect, and the equivalent of 5 c.cm. contained no pressor substance.

Urine.—Specimens of urine were collected in May and July at the same time as the blood was obtained. A further sample was tested in August. It was later decided to see whether variation in the excretion of the active substance occurred, so a series of seven one-litre specimens were collected under comparable

* Working with a Leverhulme fellowship from the Royal College of Physicians of London.
† Working with a grant from the Medical Research Council.

conditions from Sept. 8th to 20th and extracted separately.

A preliminary test on the extract obtained from the May specimen of urine showed that a considerable amount of activity was present. In the accompanying Figure the pressor action is shown and compared with that of pitressin on the same spinal cat. The types of curve given are seen to be essentially the same. Also after heating the urine extract at 100° C. at *p*H 3 for 45 minutes there was no change in activity. From this it is seen that the extract from 70 c.cm. of urine produced an 84 mm. Hg rise in pressure and is approximately the same as 0·2 unit of pitressin. In Table I the antidiuretic activity of this extract is given and may be compared with pitressin. The amount of active substance obtained from only 2 c.cm. of urine is seen to exert a definite action.



A B C D

Spinal-cat pressor assay. (A) = 0·1 unit pitressin. (B) = 0·2 unit pitressin. (C) = Extract of May urine, equivalent to 70 c.cm. (D) = Extract of May urine (equivalent of 70 c.cm.) after heating.

In Table II the assays on all the urine specimens are given. The May extract was tested on frogs by Dr. Jones and contained a melanosome-dispersing principle. Control extracts have been prepared from the urine of normal persons and tests on these have always given negative results.

The pressor substance obtained from the urine appears to have an effect indistinguishable from that in the posterior pituitary. The typical blood-pressure curve, method of extraction, and stability support such a suggestion. The difference in the amounts of pressor and antidiuretic substances suggests that they are separate principles. However, it has been shown (Dodds, Noble, Rinderknecht, and Williams 1937) that under certain conditions the antidiuretic effect of pituitary extract can be prolonged, and an apparent increase in activity be obtained. It is possible that the urine extracts described may contain material which prolongs the urinary effect but would not affect the intravenous pressor assay.

The urinary antidiuretic substance appears to differ from the vasopressive principle in that it is less stable to heat in acid solutions. In general, however, it can be seen that the active preparations from the urine behave in a way so similar to posterior pituitary extracts as to suggest that these substances are identical or closely related.

The demonstration by Gilman and Goodman (1937) that an antidiuretic substance was excreted in the urine of rats after dehydration, and that this does not happen with hypophysectomised animals, suggests that a pituitary hormone may be excreted under suitable conditions. Melville (1937) has further shown that the active substance rapidly disappears from the blood and from the observations of Dale (1909) and Heller and Urban (1935) a urinary elimination, at least in part, appears probable. Our observations that an active substance could be recovered from the urine and not the blood therefore seem in accordance with the above findings.

Although the method of extraction employed gives a low yield of active material it is apparent that this patient was excreting a comparatively high concentration of the active substance. Later assays giving lower values are in agreement with the clinical improvement that took place in this case. Further observations are in progress to determine whether active urinary preparations can be obtained in other types of pathological conditions.

TABLE I—ANTIDIURETIC ASSAY OF MAY URINE EXTRACT

No. of rats.	Substance.	Percentage of total urine excretion in—							
		½ hour.	1 hour.	1½ hours.	2 hours.	2½ hours.	3 hours.	3½ hours.	4 hours.
24	Water alone.	3·0	22·5	56·5	86·0	98·0	100·0	—	—
44	0·02 unit pitressin.	0·0	2·0	12·0	37·0	65·0	85·0	97·0	100·0
44	0·04 unit pitressin.	0·0	0·0	2·0	14·0	35·0	63·0	85·0	97·0
12	EXTRACT MAY URINE EQUIVALENT TO 2 C.CM.	0·0	0·5	3·0	12·0	33·0	61·0	86·0	95·0

TABLE II—URINE ASSAY FOR PRESSOR AND ANTIDIURETIC ACTIVITY (PITRESSIN AS STANDARD)

Urine specimen.	Pressor units per 1000 c.cm.	Antidiuretic units per 1000 c.cm.	100° C. *p*H 3 1 hr.
May	3·3	20·0	5·0
July* ..	<0·1	<0·1	—
August ..	1·17	5·0	1·3
Sept. 8th ..	1·5	2·0	<0·2
„ 10th† ..	<0·1	<0·2	—
„ 11th ..	0·1 (approx.)	<0·2	<0·2
„ 14th ..	<0·1	<0·2	—
„ 16th ..	<0·1	2·0	0·5
„ 18th ..	<0·1	<0·2	—

Pressor effect unchanged by heating.
* Urine alkaline.
† Friedman test negative.

SUMMARY

An extract containing pressor and antidiuretic activity has been obtained from the urine of a

case of suggested hyperfunction of the posterior pituitary.

The effects of this extract are similar to those produced by the pressor principle of pituitary preparations.

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ELECTROSURGICAL OBLITERATION OF THE GALL-BLADDER WITHOUT DRAINAGE*

A REPORT OF 342 CASES

BY MAX THOREK

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REMOVING the gall-bladder by means of the scalpel yields in competent hands a mortality of about 1 or 2 per cent. in uncomplicated cases. The mortality increases with age and is greater in males than in females. It ranges around 10 per cent., and taking into consideration the unreported results of the general surgeon, it may on occasion be as high as 20 or 30 per cent. or even higher depending upon the presence of complication (Table I).

An analysis of fatalities following classical cholecystectomy affords food for the following reflections:

1. From 15 to 25 per cent. of patients have enlarged bile capillaries and small bile-ducts running in the gall-bladder bed which cause bile leakage when the gall-bladder is removed from its bed with the scalpel.

2. Bile in the peritoneal cavity when sterile is a chemical poison. When infected it may give rise to biliary peritonitis.

3. A properly ligated cystic duct does not leak; bile usually issues from the gall-bladder bed as pointed out by Bakes (1928). He observed bile issuing from the wound 230 times in 346 cases of simple cholecystectomy. Like many other surgeons he was impressed by the post-operative appearance of bile in the dressing "in nearly all the cases."

4. After classical cholecystectomy the gall-bladder bed cannot always be obliterated by sutures. Drainage is then resorted to and this invites leakage. Open unprotected bile and blood channels in the gall-bladder bed, following cholecystectomy by the classical method, offer a favourable atrium for the entrance of micro-organisms.

5. Drainage causes trouble in a number of ways (augmentation of bile seepage; thrombosis and embolism; hæmorrhage from erosion of vessels; cholæmic bleeding; embarrassed cardiac action, particularly in the aged; pneumonia; acute dilatation of the stomach; biliary fistulas, &c.). To put it differently, *many patients die after gall-bladder operations not because they were not drained but because of drainage.* Nevertheless though many dangers may be eliminated by omitting drains, drainage instituted where classical cholecystectomy is practised may be the means of saving a life.

In striving to ascertain how the evils of drainage can be avoided and how one may frustrate leakage from divided capillaries and small bile-ducts, I thought that if a dry, non-leaking surface could be substituted for the discharging cavity represented by the gall-bladder bed resulting from classical cholecystectomy, it would be an important step in the right direction. Experimental studies on dogs and monkeys proved that this can be accomplished by proper electrocoagulation, which, when properly performed, converts the tissues into a hyaline-like, dry, aseptic, inert tampon. Unfortunately, electrocoagulation is still confused by the uninitiated with fulguration and carbonisation, which are entirely different in their action and effects. It is important to understand the differences (vide Ellis 1932).

Monopolar sparking from a high-frequency transformer of Tesla, d'Arsonval, or Oudin was termed *sideration* by Keating-Hart and *electrodissection* by Clark and later *fulguration* by Pozzi. Nagelschmidt introduced the now accepted term *diathermy*. *Bipolar diathermy* for surgical purposes was first called *voltaisation bipolaire* and later *electrocoagulation* by Doyen; Delherme and Laquerrière spoke of *endothermy* showing that the heat is produced in the patient's body. George Wyeth in 1926 simplified the terminology by coining the terms *medical* and *surgical diathermy*. Hanschen speaks of *exsiccation*, *charring*, Heitz-Boyer of *etincelage*, Cushing of *dehydration*, Kowarchik of *electrotomy*, von Seemen of *Schmelzschnitt*.

TABLE I

Mortality Statistics in Gall-bladder Operations (1908–32)

—	Year.	Cases.	Mortality (per cent.).
Cotte, M.	1908	Not given.	M., 33 F., 18
Davis, B. B.	1921	563	8·17
Enderlen and Hotz.	1923	12,144	9·28
Brentano.	1923	280	M., 36·78 F., 17·29
Villard.	1925	131 cholecystostomies.	M., 34 F., 21
Duclos.	1926	Global mortality.	M., 36 F., 23
Davis, B. B.	1928	160	3·75
Cattell, R. B.	1929	311 (complications 55).	M., 9·1 F., 6·8
Sanders, R. L.	1930	18 reported series, totalling 60 cases.	15
Verbrycke.	1927	169*	8·8 28·5 15
Tixier, Clavel, and Chabannes.	1932	11 males. 149 females.	M., 36·38 F., 16
Thorek.	1910–32	138 males. 511 females.	M., 16·8 F., 9·4

* Operated on by 23 surgeons; one surgeon had 10 and another had 20.

M.= Male. F.= Female.

If a flat electrode of a bipolar current is firmly applied to a tissue surface and a current of proper voltage and sufficient amperage is permitted to pass through it, dehydration and coagulation of the tissue-proteins results in a few seconds and the tissue turns white. This is *electrocoagulation*. On the other hand, if this same electrode is not applied firmly or if it is used as a unipolar instrument, a small air-space (dielectricum) intervenes between the

* Read at the British Postgraduate Medical School, London, on Sept. 8th, 1937.

electrode and tissues and *sparking*, *fulguration*, and *carbonisation* with black discoloration of the tissues results. A reapplication of the electrode to such fulgurated or carbonised surface will stop further current-penetration and prevent coagulation from taking place (Fig. 1). In cauterisation the heat is brought to the tissues from the outside by the heated instrument, whereas in bipolar or short-wave electrocoagulation the heat is produced in the patient's body (75,000 to 3,000,000 or more oscillations are produced per second depending upon the type of apparatus used).

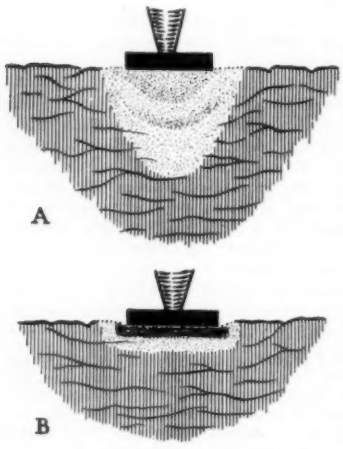

FIG. 1.—Effects of electrocoagulation and of carbonisation. In (A) one observes the degree and extent of electrocoagulation when correct technique is used. The dense area immediately under the electrode indicates that here the action is of greatest intensity. It gradually decreases as indicated in the diagram. Marginal action is seen to extend for some distance from the border of the electrode. With improper use (dielectricum) and the application of too much current charring results immediately under the electrode (B). Such eschar insulates the area acted upon and further effects of coagulation are frustrated.

Definite tissue changes take place when the liver is subjected to the electrocoagulating bipolar current. This problem has been ably worked out jointly by Kuntzen and Vogel (1931). Electrocoagulated surfaces within the abdomen do not slough as do surfaces on the exterior or in open cavities (mouth, vagina, cervix); in such places coagulated areas are eliminated by extrusion, but within the abdomen they become encapsulated and are absorbed. Electrocoagulated surfaces on parenchymatous organs develop an affinity (a positive chemotaxis) for serous surfaces attracting contiguous organs covered with serosa (omentum, stomach, bowels). I have observed such agglutinations three hours after electrocoagulating the surface of the liver. Firm union takes place in a comparatively short time. Capillaries and blood-vessels of an electrocoagulated area do not thrombose but a recession of the column of blood is formed within the vessel, above and below the point of contact of the electrode, and the vessel walls coalesce with the contiguous, homogeneous, hyaline-like structures mentioned. Such changes do not take place with fulguration or carbonisation.

Another important fact to be remembered is that electrocoagulation of the tissues in the gall-bladder bed frustrates secondary hæmorrhage because of the pressure exerted by the sequestrated, coagulated segment against the contiguous tissue. Thus *electrocoagulation substitutes a secure sterile tampon for an insecure open cavity at the site of the gall-bladder bed*, resulting from classical cholecystectomy. Of late I have been using short-wave unipolar coagulation with entire satisfaction.

EVOLUTION OF AUTHOR'S METHOD

For some years we performed experiments upon large dogs and monkeys (*Macacus rhesus* and *M. leoninus*). Anatomic differences and lack of gall-bladder pathology in these animals should be kept in mind. My experiments allow the following conclusions:—

1. The posterior gall-bladder wall and bed of the gall-bladder may be effectually electrocoagulated and their structure converted into an inert dehydrated hyaline-like aseptic tissue.

2. Electrocoagulated borders of the gall-bladder thus treated may be approximated with sutures.

3. The ligamentum falciforme hepatis may be completely detached and when superimposed over an electrocoagulated surface will become intimately united with it.

4. The abdomen may be closed without drainage in such instances provided the cystic duct is securely ligated and the cystic artery closed by double ligation.

Priority for introducing electrosurgery in gall-bladder disease belongs to Trowbridge. He described electrosurgical cholecystectomy with recovery in 1928. Heymann (1930) substituted the diatherm knife for the scalpel, as did also Keysser, Mirizzi, and others; the results were little different from those of classical cholecystectomy. Pribram (1928, 1929) used carbonisation of the mucosa with the Paquelin burner and later (1933) with a diatherm apparatus. He called his method *mucoclasis*. Pribram's procedure is a fulguration method aiming at destruction of the mucous membrane of the gall-bladder; but as I have shown in another communication (1933) it does not entirely obliterate the gall-bladder and thus eradicate disease, for the charred mucosa prevents the current from reaching the depths of the affected zone in the gall-bladder wall—viz., the Rokitansky-Aschoff sinuses which harbour infection Equally, simple fulguration is not adequate where the infection has extended into the depths of the gall-bladder wall, bed and liver substance to various depths. Whitaker (1930) combined Pribram's cauterisation procedure with drainage in 16 cases, disregarding the admonition that electrocoagulated surfaces must never be drained, and reported a mortality of 18·75 per cent.!

After using Pribram's method for some time, I found that patients were not relieved of their symptoms and required reoperation. At such time it was noted that there was regeneration of the mucous membrane of the gall-bladder mucosa and the

TABLE II

Electrosurgical Obliteration of the Gall-bladder

Cases operated by Thorek		267	
Cases operated by others (Kellogg, Warren, Finlayson, Baller, de Victoria, Weyman, Heineck, Montgomery)		75	
Total of unselected cases of gall-bladder disease operated on to date (August, 1937)		—	342
Sex	Male	43	
	Female	299	
Age	Youngest	18 years	
	Oldest	74 "	
Average high temperature		100° F.	
Average days in hospital		15·5	
Pathologic conditions found at operations—(1) acute and chronic cholecystitis; (2) cholelithiasis; (3) hydrops vesicæ felleæ; (4) empyema of the gall-bladder; (5) acute hæmorrhagic cholecystitis; (6) hepatitis; (7) adhesions; (8) chronic pancreatitis.			
Post-operative complications—(1) bilateral pleurisy with effusion, ten days after operation; cleared up; patient left hospital 27 days after operation; (2) wound infections (3 cases).		About 0·5 %	
Mortality (three deaths). Causes of death—(1) bilateral pulmonary collapse; (2) pneumonia, five weeks after operation; (3) subdiaphragmatic abscess.		About 0·5 %*	

* Since this paper was presented 9 additional cases have been operated on by me by the method described without a death, making a total of 351 cases.

diseased layers of the gall-bladder were little or not affected at all. Infection persisted. It occurred to me then that complete destruction of the gall-bladder wall would be a step in the right direction. The following procedure was evolved :—

(1) Electrosurgical obliteration of the posterior gall-bladder wall and gall-bladder bed by electrocoagulation (biterminal or by short wave) thus substituting a sterile, dehydrated tampon for a bleeding, bile-leaking surface.

(2) The great tendency of electrocoagulated surfaces to become agglutinated with serous surfaces caused me to muster the falciform ligament into service.

(3) Strict avoidance of drainage.

So far (August, 1937) 342 operations have been performed by my method in cases of gall-bladder disease including gangrene, empyema, and sclerosis. Of these, 267 cases were done by myself and the remainder by Drs. Homer S. Warren, Jr., Bliss Finlayson, Edward L. Kellog, Cassius Lopez de Victoria, Stanley Baller, and others. There were 3 deaths in this series. One patient succumbed to an undiagnosed chronic subdiaphragmatic abscess some time after operation (post-mortem confirmation). Another patient died from massive bilateral pulmonary collapse, and the third from pneumonia about three weeks after the operation. If these 3 deaths are credited to the operation, we are confronted with a mortality of less than *1 per cent.* A comparison of these results (Table II) with those shown in Table I shows a sharp contrast. Whether my procedure alone or the painstaking pre- and post-operative care of patients, or both, was responsible for these results, I cannot say; but certainly thorough preoperative preparation of the patient is imperative.

OPERATIVE TECHNIQUE

STEP 1.—In well-trained hands, subarachnoid block is the anæsthetic of choice; otherwise, inhalation anæsthesia is administered. The patient is placed in the Mayo-Robson position with the lower thorax and upper abdomen sufficiently elevated. When the biterminal apparatus is used, a large flat, indifferent electrode is snugly applied over the sacrum. Where short-wave is used an indifferent electrode is not necessary. Preferably a straight longitudinal paramedian incision is made beginning at the lower costal margin and extending to about the level of the umbilicus. Exposure must be thorough.

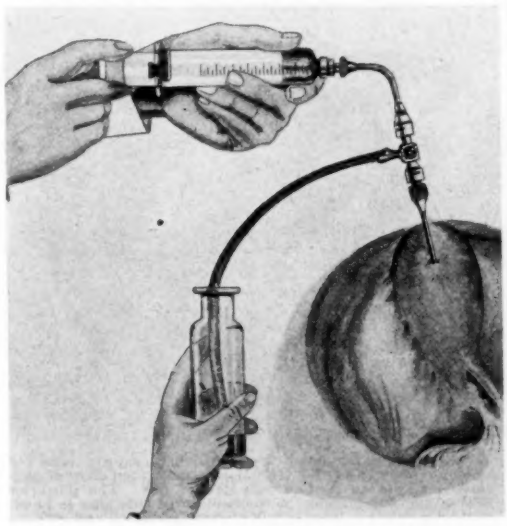

FIG. 2.—Author's gall-bladder aspirator.

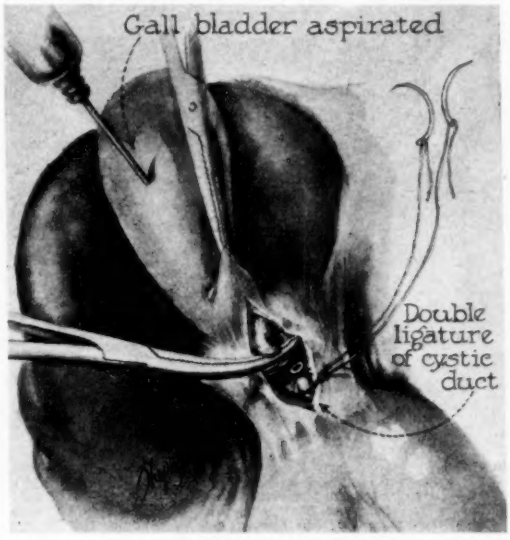

FIG. 3.—After aspiration of the gall-bladder the cystic duct and cystic artery are doubly ligated, the duct and artery divided and the ligature cut short.

STEP 2. *Mobilisation of the falciform ligament.*—This structure is completely detached from the anterior abdominal wall. The detached ligament is now placed in a receptacle containing warm normal salt solution. In detaching the falciform ligament from the anterior abdominal wall, some small vessels are severed; these must be carefully ligated. Pack the viscera out of the way and obtain good exposure by efficient retraction. (The hypodermic injection of an ampoule of Pitressin about ten minutes before the operation is begun aids in keeping the bowels out of the way.)

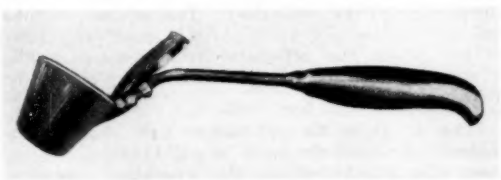

FIG. 4.—Author's non-spillable bile receptacle.

STEP 3. *Exploration of biliary passages.*—I cannot agree with some writers that opening of the common duct should be resorted to as a routine or that instrumental exploration of the common duct is free from danger. The gall-bladder is aspirated. I use an especially designed aspirator which permits evacuation of the gall-bladder contents into a receptable via a rubber tube (Fig. 2). After aspiration of its contents, the gall-bladder is filled with hexylresorcinol (S.T. 37) or some other antiseptic solution. Only about 5 per cent. of bile contains micro-organisms,

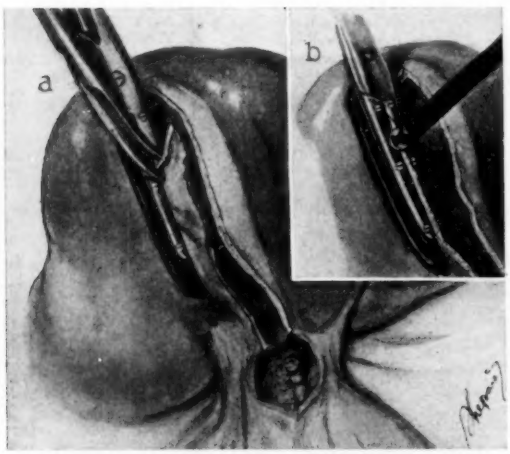

FIG. 5.—(*a*) Opened gall-bladder crushed with author's insulated angiotribe forceps and redundant portion of gall-bladder removed with ordinary scissors. (*b*) Remaining strip of gall-bladder in grasp of forceps being electrocoagulated with short-wave current. The current passes through the forceps but not beyond the insulated protector.

while their presence in the gall-bladder wall may be demonstrated in over 70 per cent. of cases. For that reason I introduce an antiseptic into the gall-bladder before opening it. If the cystic duct is patent we probe the ducts; otherwise digital exploration is resorted to. The extrahepatic biliary passages may be explored through the opened gall-bladder. The cystic artery and duct are ligated doubly and jointly, cutting both ligatures short. Experience has taught me not to attempt extensive dissections in search for the cystic artery if it is not easily isolated, but to splint it with the ligature to the cystic duct. This advice could be followed particularly where there is much infiltration in and about the hepatoduodenal ligament. The frequently abnormal course of the artery should be kept in mind (Fig. 3).

STEP 4. *Open the gall-bladder and evacuate its contents.*—I have designed a gall-bladder content receptacle which collects the evacuated material without spilling it (Fig. 4). Old-fashioned gall-bladder spoons are too shallow and offer no protection against contamination from spilled contents. My instrument consists of a cone-shaped container measuring 2¼ in. in diameter and 2 in. in depth, mounted on a shaft of suitable size. A close-fitting cover is hinged and held open, when in use, by means of spring friction clips. Slight pressure upon the cover releases it so that the material cannot spill during its removal. The front part of the receptacle is somewhat concave, allowing sufficient latitude for proper contact with the subcholecystic structures, and its edges are rodded, thus avoiding injury to the liver.

STEP 5.—*Split the gall-bladder* longitudinally from above downward with an ordinary pair of scissors. Grasp half of the gall-bladder wall with an insulated angiotribe forceps or a rubber-covered ordinary artery forceps, of proper size and thickness. In closing the forceps, the gall-bladder wall is crushed and the blood-vessels coursing in it closed by compression. With an ordinary pair of scissors remove the redundant portion of the gall-bladder wall. Apply a small electrode firmly along the exposed margin of the gall-bladder held in the forceps. The smaller the electrode, the quicker the action. The current will electrocoagulate the gall-bladder wall in the grasp of the forceps. After sufficient coagulation, the forceps is opened; a compressed coagulated ribbon of tissue remains (Fig. 5A and B). Repeat the procedure on the other half of the gall-bladder. Electrocoagulate the remaining portion of the posterior wall of the attached gall-bladder, which has not yet been acted upon (Fig. 6).

In my early work I thought it advisable to electrocoagulate slowly but experience has taught me that quick decisive coagulation with firm electrode contact yielded the best results. I use a current of low voltage and high amperage on a rather small electrode. On this unit (Fischer Model S.W. 12) as the voltage drops, the amperage increases; this affords prompt, thorough dehydration and a sharply defined line of demarcation between the coagulated and non-coagulated tissue. Tissue thus coagulated will retain less moisture than tissue coagulated more slowly and less heat is radiated to the contiguous structures. Another reason for substituting rapid coagulation with a smaller electrode is that slow coagulation results in heating of the tissues to quite an extent beyond the electrode, about 0·5 cm. to each millimetre. There is no sharp line of demarcation in slow coagulation; the tissue changes are gradual.

It must again be emphasised that the electrode must be applied snugly to avoid carbonisation. The current should be turned on only after the electrode is firmly in contact with the tissues to be acted upon, and that fulguration and its attendant carbonisation must be avoided because such carbonisation will defeat the purpose of the operation.

When the steps of the operation are completed as here outlined, there will be present an area represented by the electrocoagulated posterior wall of the gall-bladder attached to the gall-bladder bed.

STEP 6.—*Approximate the electrocoagulated edges of the gall-bladder* by a few interrupted or a continuous suture of fine catgut. While thus approximating the edges without strangulating them, the needle carrying the catgut must not penetrate the liver substance—only the edges of the coagulated ribbon are picked up (Fig. 6D).

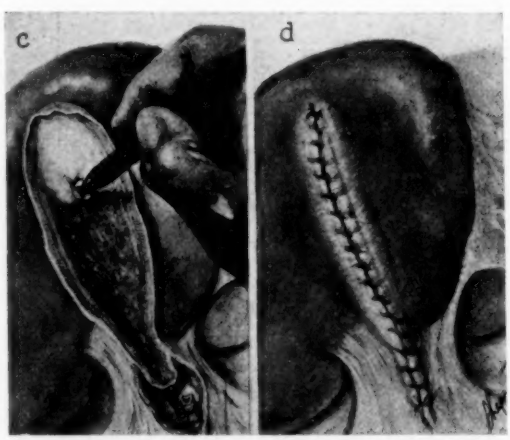

FIG. 6.—(*c*) The insulated angiotribe forceps are removed leaving ribbon of crushed and electrocoagulated strip of gall-bladder tissue all around gall-bladder bed. The posterior wall of the gall-bladder in the middle of this ring is being electrocoagulated. (*d*) Strip of electrocoagulated and crushed ring of tissue of gall-bladder united with continuous catgut sutures.

STEP 7. *The detached falciform ligament is now removed from the salt-solution container.*—One end of the ligament is sutured to the upper end of the united, coagulated gall-bladder bed (Figs. 7 and 8). The lower third of the ligament is now stitched to the lowermost end of the approximated electrocoagulated surface and the free end of the ligament is placed, but not sutured, against the doubly ligated end of the cystic duct and artery. An effective serous covering is thus formed over the gall-bladder bed and cystic duct. Two sutures are used as a rule, one above and one below.

STEP 8.—*The laparotomy packs and retractors are removed;* the field of operation is dried and the abdominal wall closed in the usual manner *without drainage.*

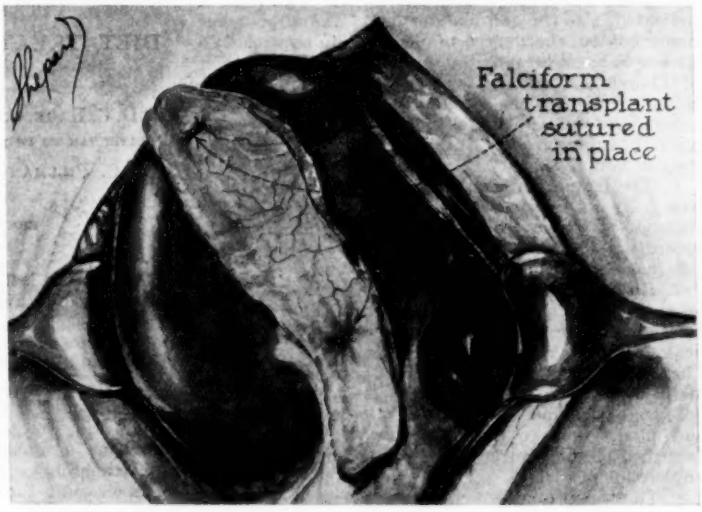

FIG. 8.—Appearance of field of operation after completion of final stage.

What becomes of the structures in the gall-bladder bed after the operation described? The answer to this has been given based upon animal experiment. Recently a post-mortem examination was done on a patient who died of acute pulmonary tuberculosis on whom I performed this operation eighteen months before her last illness. Microscopic sections showed firm union of the ligament to the underlying liver, the cords of which appeared histologically normal.

CONCLUSIONS

1. A method of electrosurgical obliteration of the gall-bladder is described which when carefully followed may be used in simple and complicated cases of gall-bladder disease without resorting to drainage. It reduces mortality to a minimum and cuts hospitalisation. The much dreaded age factor in these operations loses its terror. Shock is usually absent.

2. Failures and fatalities in classic cholecystectomy after the operation are often due to bile leakage, as a result of an inability to obliterate and cover the gall-bladder bed, which contains bile capillaries and often larger bile-ducts, in 15 to 25 per cent. of the cases. Drains invite bile seepage. The method here described seals these openings by electrocoagulation. A sterile, hyaline, dry, protective layer is substituted for a raw unprotected surface. The falciform ligament is superimposed over this area. Drainage is entirely omitted.

3. The operation here described must not be confused with mucoclasis practised with the cautery or with "diatherm-fulguration" which aims at burning or carbonisation of the mucous membrane of the gall-bladder, or with so-called "electrical" or "electrocholecystectomy" where, instead of the scalpel, the diatherm cutting knife is used.

4. A prerequisite to the achievement of satisfactory results is a patent common duct. An occluded cystic duct from any cause is an indication for this operation.

5. Experimental studies have shown that any method of carbonisation, such as fulguration, Paquelin, and electrocauterisation predisposes to hæmorrhage, thrombosis, and embolism. Such cauterisation will not destroy pathogenic micro-organisms in the depths of the affected gall-bladder wall. The operation here described is free from these drawbacks and effectually accomplishes destruction of

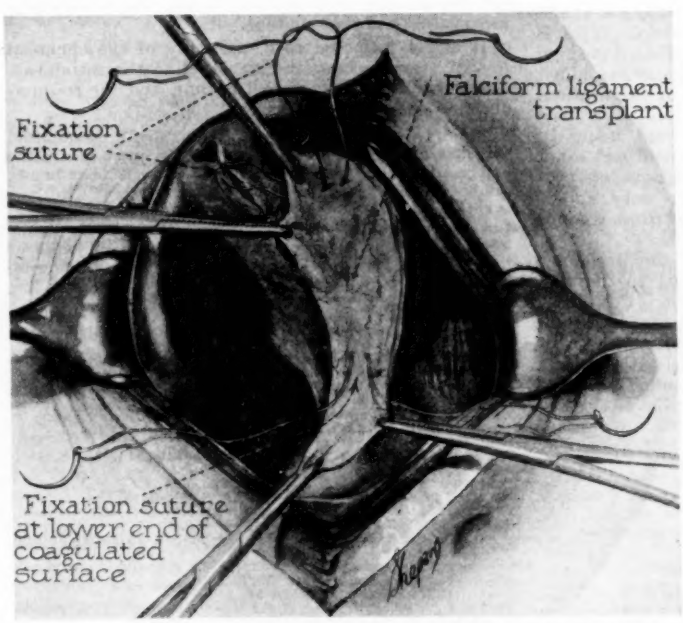

FIG. 7.—Note borders of the electrocoagulated area approximated with fixation suture. Additional interrupted sutures of fine catgut approximate the borders of the electrocoagulated zone along its entire extent. The mobilised falciform ligament is fixed over the closed electrocoagulated surface. Note placement of sutures.

the entire thickness of the gall-bladder wall and, if indicated, also the gall-bladder bed. The surgeon has under control the degree of penetration he wishes to accomplish. Whitaker (1935) states: "The method of Thorek emphasizing contact coagulation to considerable depth seems preferable to fulguration. . . . I am grateful to Thorek for the distinction and wish to emphasize the necessity for real contact coagulation. . . ."

6. The ligamentum falciforme hepatis is used as a free graft, its serous structure covers sutured or raw surfaces to great advantage, thus reinforcing and protecting the areas concerned against seepage and safeguarding the processes of repair.

7. Electrocoagulated areas of intra-abdominal organs tend to heal by encapsulation. They do not interfere with wound healing. On the contrary, they heal promptly when the wound is closed securely. Therefore drainage is not only undesirable but is distinctly deleterious. Many patients succumb because of drainage. It prolongs the healing process and predisposes to complications. The operation described eliminates the necessity of drainage and its unpleasant sequelæ.

8. The thesis here presented deals with diseases of the gall-bladder. It must be stated, however, that where choledochostomy or hepaticostomy is indicated, these may be done, in conjunction with my procedure. Experimental studies showed that the implanted falciform ligament over the obliterated gall-bladder bed forms sufficient protection and does not interfere with a Kehr, Orr or similar drainage-tube.

9. Whereas the global mortality of removal of the gall-bladder by the scalpel method in complicated cases still ranges around 10 per cent. or more, the mortality in the series of 342 unselected cases here reported was about one-half of 1 per cent.

10. Electrosurgical cholecystectomy or electrocholecystectomy, so-called, is an entirely different procedure; it is nothing more than classical cholecystectomy in which the electric cutting knife is substituted for the scalpel. It has contributed nothing to the improvement of the old method and has added the danger of explosion by sparking or fulguration. Drainage adds to its drawbacks.

11. Many decades ago attempts were made to destroy the gall-bladder with carbolic acid and other escharotic substances. This is an antiquated procedure and quite ineffective, acting only superficially, demanding prolonged periods of drainage and often leading to fistulisation.

12. A thorough understanding of the principles involved in this technique are essential to the successful performance of the operation here described. The tyro or the inexperienced cannot expect results obtained by serious workers. In speaking of electrosurgical resection of the prostate J. McCarthy recalls that it is not a "procedure for amateurs" or to be practised as "a hobby." The same applies to electrosurgical obliteration of the gall-bladder. It exacts careful technique and meticulous attention to detail.

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VITAMIN-C OUTPUT IN DIET TREATMENT OF RHEUMATOID ARTHRITIS

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DURING the last 18 months we have been studying the effects of a raw vegetable and fruit diet on various rheumatic conditions. The results of the therapeutic trial have already been reported by one of us (Hare 1936).

The description of the diet as an unsalted, raw, vegetable and fruit régime is not really accurate, since during the period of strict dieting each patient received, in addition to the fruit and vegetables, approximately a pint of milk, 3 oz. cream, 2 oz. olive oil, and ¾ oz. of uncooked rolled oats a day. Later, as an intermediate diet, the animal protein was increased by the addition of 2 eggs, 1½ oz. cheese, and 2 oz. meat *or* fish daily; no bacon or salted meat was given. Even then the diet differs from an ordinary mixed diet by the very high proportion of uncooked fruit and vegetable and the negligible amount of sugar and all forms of cereals. For details of the diet, the reader is referred to the original paper (1936).

CASE REPORTS

Biochemical tests were carried out in 6 cases of rheumatoid arthritis with a view to determining the effective factor in the diet. In this the two most striking variants from an ordinary mixed diet were the abnormally high vitamin C and the abnormally low sodium-chloride content.

It is not proposed to discuss any of the chemical investigations except those related to the estimation of the vitamin-C intake and output, and the sodium-chloride output.

CASE 1.—A man, aged 28, with slight rheumatoid arthritis, affecting chiefly feet and hands, onset 12 months before admission. Had lived in damp new cement house, unable to feed himself properly recently.

On admission—(May 28th, 1937). No evidence of general toxæmia except loss of weight. Very little visible deformity, but slight rheumatoid changes in small joints of hands, spindle-shaped swelling on 2nd and 3rd fingers,

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and feet which were hot and painful. Walking painful. Septic tonsils.

Progress.—Occasional temperature of 99° F. in first three weeks of treatment. June 11th: strict diet. June 25th: intermediate diet. Slow improvement. (Chart 1.)

On discharge (July 28th).—There was no swelling, redness, or œdema of any joint. Grip strong. Some stiffness of left elbow and shoulders.

CASE 2.—Man, aged 45, who had slight rheumatoid arthritis, chiefly affecting the small joints of the hands. Onset two months before admission with pain in joints and fever. He had lost weight (very tall and very thin). History of rheumatic endocarditis.

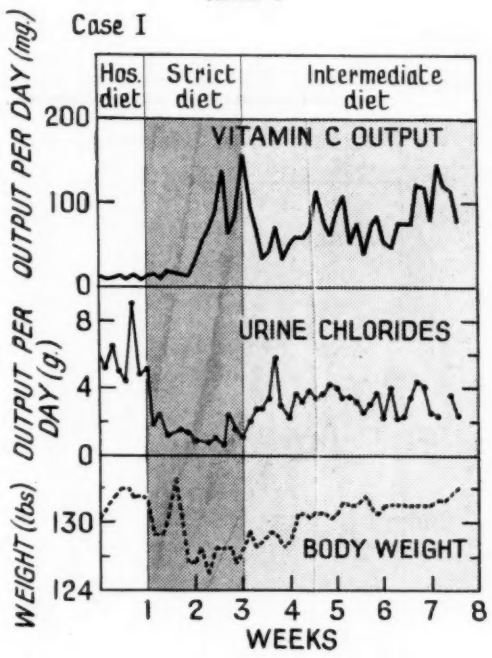
On admission (March 19th, 1937).—No evidence of general toxæmia except loss of weight. No rise of temperature or pulse-rate. Slight peri-articular swelling of metacarpophalangeal and phalangeal joints, no gross deformity. Wrists, slight swelling and painful. Elbows stiff. Knee and feet movements good, no swelling or pain. Old rheumatic valvular lesion. Mitral stenosis. Blood pressure 110/58 mm. Hg.

Progress.—April 1st: put on ascorbic acid, 400 mg. a day, and vitamin A. April 14th: strict diet added. April 24th: pains varying from day to day. April 28th: intermediate diet. May 12th: improved; feels very much better. (Chart 2.)

On discharge (July 22nd).—Walking, all movements without pain. Grip much improved. To continue diet. Sept. 5th: keeping well, still on diet; has returned to work.

CASE 3.—Man, aged 28, with synovitis of left knee in March, 1937, and November, 1937. Tonsillectomy and tooth extractions in March with some improvement, but pain and swelling returned in knee and also pain in hips and sacro-iliac joints.

CHART 1



BLOOD CHEMISTRY

Weeks	1	2	3	4	5	8
R.B.C. (millions)	4·465	5·250	5·125	4·525	4·875	4·610
Cell volume (cubic μ)	82·80	82·00	81·80	83·90	83·00	82·30
Uric acid (mg. per 100 c.cm.)	3·47	4·14	3·73	3·32	2·50	2·54
Phosphorus („)	3·19	3·45	3·65	3·74	3·68	3·50
Calcium („)	11·10	11·55	10·70	10·30	10·60	11·25
Alkali reserve (c.cm. per 100 c.cm.) %	65·80	67·40	69·12	68·02	68·50	71·54
Sodium (mg. per 100 c.cm.)	325·0	325·0	325·5	324·0	325·0	325·0
Chlorides („)	339·3	339·3	348·9	356·0	347·7	356·5

On admission (June 22nd, 1937).—Poor, toxæmic looking. On the dole—wife and 2 children. Left knee swollen. Tender over both sacro-iliac joints. All movements of hip-joint limited by pain. Blood pressure 135/70 mm. Hg. Definite muscular rheumatism of trapezium, lumbar and gluteal muscles.

Progress.—June 23rd: knee-joint aspirated. July 9th: ascorbic acid, 400 mg. daily. July 21st: diet + ascorbic acid, 400 mg. daily. Slight improvement on diet; walking with ease.

Discharged himself, owing to loss of unemployment pay while in hospital.

CASE 4.—Man, aged 52, referred from Red Cross Clinic. Invalided out of the Army in 1917 for nephritis, said to have had no recurrence. He had suffered from rheumatism for three years, and had been continuously under treatment. Three periods of residential treatment, including Bath, Bognor, and Southport. He had vaccine, electrical treatment, radiant heat, &c., and was treated for positive complement-fixation test. He had been unable to work for 8 months before admission. The rheumatism was both muscular and arthritic. There was severe muscular stiffness and pain, especially of the neck and shoulder muscles. The joints affected were the wrists and small joints of the hands. There was but little swelling or deformity. X ray showed some atrophic arthritis of hands, left shoulder, and hips. There was slight disability of the hands, chiefly due to weakness, and gait was slow and stiff.

On admission.—Arteries tortuous and hardened; blood pressure 195/128 mm. Hg, 170/120 after a week's rest. No wasting or anæmia or rises of temperature or other evidence of general toxæmia. Later he complained of pain in one groin associated with enlarged glands. This was persistent and hindered his walking. Seen by surgeon who advised no interference.

Progress.—Jan. 26th: ascorbic acid, 400 mg. daily. March 10th: strict diet and sodium chloride capsules, 5 g. March 23rd: strict diet, no salt; good improvement. April 7th: intermediate diet.

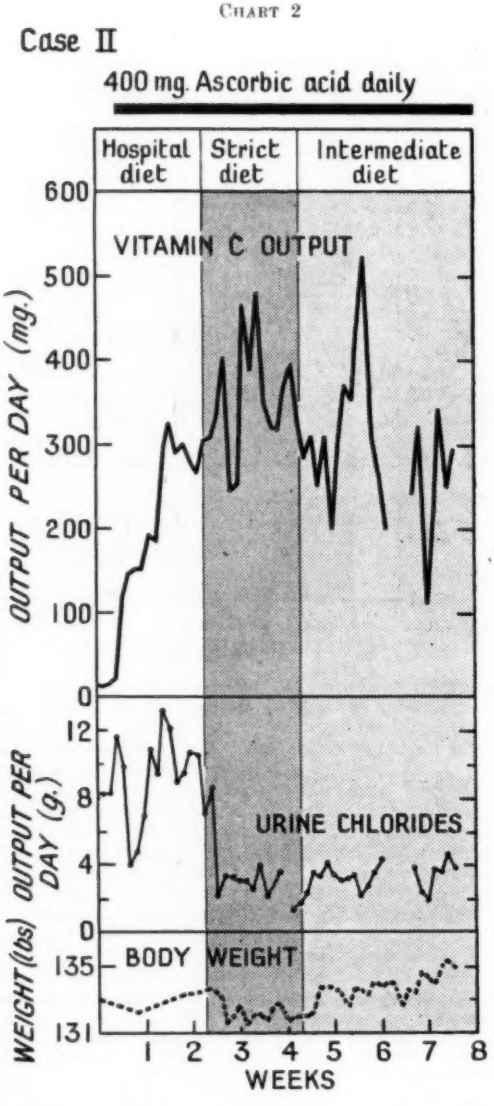
On discharge (May 7th).—Gait normal. No acute swelling of any joints; some painful gland in right groin. To continue diet at home.

Readmission 5 weeks later (June 28th) with severe relapse. He had been cutting wood, washing paint, &c., and had *not* kept to diet. Gait very painful. Swelling of metacarpophalangeal joints, phalangeal joints and ankle-joints. Pain in knees. Blood pressure 200/137 mm. Hg. Progress on diet good, but he was troubled with painful glands in right groin.

Discharged (July 29th), once again free from signs of active disease.

CASE 5.—Man aged 43. Severe progressive rheumatoid arthritis, with muscular pain, stiffness, and much wasting.

CHART 2



knees, ankles, and feet. Rheumatic nodules on elbows. Unable to move hands and feet on account of pain and stiffness. Enlarged and tender inguinal glands.

Radiograms show osteo-arthritic changes. Condition in metatarsal phalangeal joint suggests gout. Blood pressure 100/60 mm. Hg.

Progress.—He was in hospital 6 weeks on other treatment without improvement before diet was tried. Enlarged tonsils and sore-throat developed within 10 days of admission. April 23rd: ascorbic acid, 400 mg. daily; hospital diet. May 7th: strict diet with 15 g. sodium chloride and ascorbic acid. May 28th: strict diet without salt, with ascorbic acid. June 11th: intermediate diet. (Chart 3.)

On discharge.—Condition much improved. August 3rd: tonsillectomy; no exacerbation of rheumatic symptoms.

CASE 6.—Man, aged 67, with multiple arthritis, coming on in July, 1936, after getting wet through. Health had been good until 2 or 3 years previously. Aching pain in chest radiating to arm and flatulence in 1933.

On admission.—No evidence of toxæmia. Pain and stiffness of joints. Afebrile from 3–10 days in hospital. Radiograms: severe osteo-arthritis lower cervical spine; all joints of hands, acromio-clavicular joints, and knees show some changes. Wassermann reaction and Kahn negative. Blood pressure 110/86 mm. Hg.

Progress.—Movements more limited and joints more painful before any treatment started. Temperature occasionally 99° F. April 13th: ascorbic acid, 400 mg. daily. April 19th: joints became stiffer and more painful. April 30th: diet I cooked and vitamin C; worse, effusion in left knee. May 14th: strict rheumatic diet; for the first week movement of joints improved and painless. May 17th: warmed milk added. May 20th: joints swollen. May 28th: intermediate diet with 1½ pints of milk given for one week. Effusion in olecranon bursa. Stiffness and pain increased. Febrile 99°–100° F.

He became on the whole progressively worse, but it was impossible to keep him strictly to the diet because of the condition of his mouth. (Tongue red, dry, and fissured.)

COMMENTS

All 6 cases were observed during a preliminary period when they were on the usual full hospital diet. After this Case 1 was given the strict raw vegetable and fruit diet for 2 weeks, followed by 5 weeks on the intermediate, and no extra ascorbic acid was added. All the other cases had hospital diet + vitamin C before further dietetic changes.

Cases 2 and 3 were given ascorbic acid, 400 mg. per day throughout the diet changes. Case 3 discharged himself after 2½ weeks owing to financial difficulties at home. Case 4 was given ascorbic acid, 400 mg. daily, until the diet was started. He was kept on the strict diet 4 weeks. For the first 2 weeks sodium chloride was added, and for the 3rd and 4th weeks he had strict diet only, and then went on to the intermediate diet for 4 weeks. Case 5

Weeks	1	2	3	4	5	8	11
R.B.C. (millions)	3·810	4·010	3·975	4·380	4·485	5·200	5·210
Cell volume (cubic μ)	89·80	92·20	93·00	90·20	89·30	87·90	88·90
Uric acid (mg. per 100 c.cm.)	3·74	2·63	2·72	3·98	4·34	3·77	4·72
Phosphorus („)	3·45	3·25	3·85	3·79	3·79	3·36	3·70
Calcium („)	9·40	12·35	11·40	11·45	11·20	11·70	9·80
Alkali reserve (c.cm. per 100 c.cm.) %	69·55	72·30	71·96	69·90	—	69·40	73·42
Sodium (mg. per 100 c.cm.)	325·5	325·0	326·0	327·0	325·0	324·0	323·8
Chlorides („)	363·7	363·7	377·4	340·2	362·2	357·3	343·2

This began 12 months before admission—unable to work for 12 months.

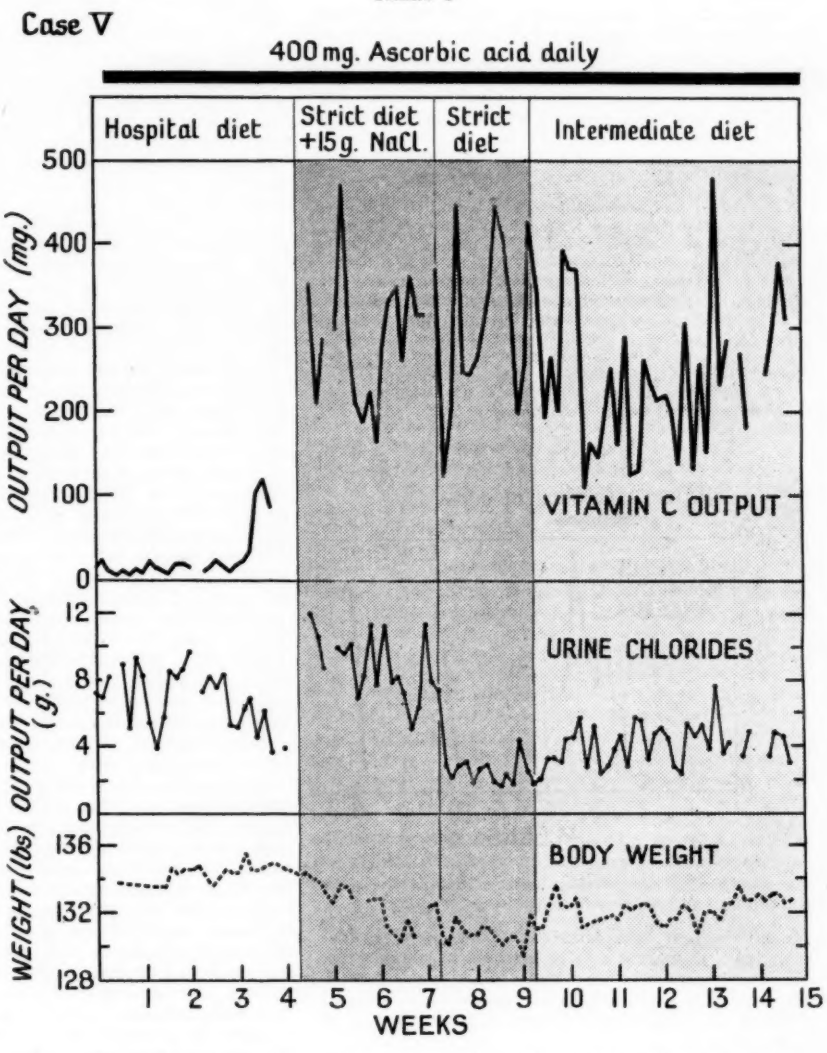
On admission (March 26th, 1937).—Patient confined to bed. Very emaciated. Temperature normal. Pulse-rate normal. Hæmoglobin 78 per cent. Joints affected: shoulders, elbows, wrists, and small joints of hands, also

was given ascorbic acid throughout, and since he had, while in hospital, a streptococcal throat infection with ear involvement, relieved by Prontosil treatment, he was also given vitamin A. The first 2 weeks on strict diet, he had added sodium chloride, followed by 2 weeks without added sodium chloride before proceeding to the

intermediate diet, which he was given for 6 weeks. Case 6 was given ascorbic acid throughout, and here the strict diet was cooked. No capsules of sodium chloride were given, but latterly traces were allowed as the diet otherwise was so unpalatable. This cooked strict diet was followed by 2 weeks uncooked strict diet. He had trouble with mouth and teeth, and was given throughout extra milk. Throughout the time of investigation he had shown an evening temperature of 99°–99·4° F. for three or four evenings a week. When the diet was increased (intermediate diet) his temperature remained for 4 days between 99° and 100° F., so the investigations were stopped and the patient put on a febrile diet. This case was deemed a failure, because it was not possible to keep to the diet.

Output of vitamin C in the urine during all these periods was estimated daily* (Johnson and Zilva 1934). The intake was assumed to be approximately that present in the fruits and fruit juices and roots taken. The ascorbic acid content of each was estimated, and these values used throughout the tests. During the preliminary period on the hospital diet all 6 cases had a very low vitamin-C output (see Charts for details of Cases 1, 2, and 5), the highest daily average being only 14·9 mg., and the lowest 2·22 mg. per diem. One patient with an average daily output of 12·7 mg. had been under treatment in hospital for 4 weeks before the tests were started. According to the usually accepted views (Schultzer 1936, Archer and Graham 1936) these figures indicate a condition of incipient scurvy. The excretion of ascorbic acid rose in all the cases receiving 400 mg. of ascorbic acid from the daily average of under 15 mg. to the maximum value ranging from 200–320 mg., but the length of time

CHART 3



Weeks	1	2	3	4	5	6	7	8	9	10	11	12	14
R.B.C. (millions)	5·070	4·940	4·810	4·790	4·820	4·950	4·830	4·660	4·820	4·835	4·859	4·920	5·005
Cell volume (cubic μ) ..	80·00	82·00	83·50	85·00	80·20	81·00	83·00	83·40	83·00	80·80	80·20	80·20	81·50
Uric acid (mg. per 100 c.cm.)	1·70	1·92	3·19	2·45	3·08	2·70	2·62	2·88	2·31	3·16	3·01	2·67	2·14
Phosphorus (mg. per 100 c.cm.)	3·33	3·80	3·68	3·68	3·90	3·27	3·40	3·36	3·85	3·80	3·32	3·67	3·70
Calcium (mg. per 100 c.cm.)	10·30	10·20	10·70	10·80	10·80	10·20	9·30	10·30	9·10	9·20	12·40	10·60	10·20
Alkali reserve (c.cm. per 100 c.cm.) %	—	66·20	58·90	61·65	59·50	—	58·90	60·00	65·80	65·76	66·10	64·82	66·26
Sodium (mg. per 100 c.cm.)	328·6	329·2	328·9	327·9	329·5	—	329·9	328·9	328·0	327·4	327·7	328·3	328·0
Chlorides (mg. per 100 c.cm.)	352·3	359·1	359·1	339·1	362·2	—	366·1	356·2	351·3	345·8	339·3	352·1	347·7

* Each sample was titrated as soon as it was obtained.

before these high values were reached varied greatly from case to case.

In Cases 3, 5, and 6 it took 4, 5, and 8 days respectively before the excretion rose above the prescorbutic level of 20 mg. per day, and even after that the rise was slow, indicating a severe degree of desaturation. In Cases 2 and 4 the output rose on the 1st and 2nd day, and both cases reached a much higher level.

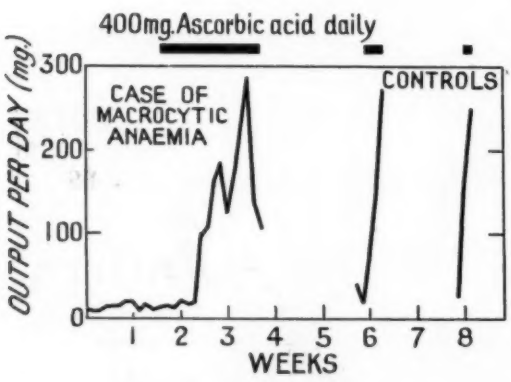
The delay in excretion in Cases 3, 5, and 6 may have been partly caused by the presence of recent septic foci.

Case 5 had had a recent attack of tonsillitis and piles, but in the other two cases no septic foci could be found.

Case 1, treated with diet only, took 8 days to show any increased excretion of ascorbic acid, and took a further 5 days before the excretion was above 100 mg. in the day. The highest value reached was 165 mg. on the 14th day of strict diet, and on the intermediate diet the values fell at first, finally, after 5½ weeks, to reach a level of 80–140 mg. per day, the approximate ascorbic acid content of the diet being 116–160 mg. per day.

It is interesting to note that Case 4, who was given the ascorbic acid for 2 weeks only, previous to the strict diet,

CHART 4



became balanced on the intermediate diet at the same level (80–140 mg. per day) during the 1st week.

In all the four cases that were given ascorbic acid throughout, there was a large increase in the ascorbic acid output directly the strict diet was started. This would be expected if the patients had become saturated by the preliminary administration of ascorbic acid.

The sodium values follow the chloride values very closely and the total output was estimated in all the patients except Case 3, and it will be seen from the charts that the chloride output falls to approximately 2 g. a day within 48 hours, and remains low throughout the strict diet to rise a little on the intermediate diet—i.e., with the addition of meat, egg, and bread in small quantities.

Controls.—To find healthy controls in hospital is difficult, and one patient tested had a macrocytic anæmia and had been in hospital many weeks (Chart 4).

During this time, every conceivable type of treatment for his anæmia had been tried without success. His vitamin-C output is shown (Chart 4), and it will be seen that the excretion did not rise until after 7 days, but before this, the blood count started to rise, and the patient's condition rapidly improved. On questioning him afterwards, it was found that he had been put on a diet by his doctor outside to cure his indigestion, and the diet lacked any food containing vitamin C.

The other two controls whose charts are shown (Chart 4), are those of healthy men living in hospital.

DISCUSSION

The average urinary excretion of normal adults on a good mixed diet appears to be about 30 mg. per day. On the hospital diet and presumably on the diet of the poorer income groups of the population, the daily excretion is below 15 mg., a value usually accepted as suboptimal and indicating a condition of incipient scurvy. From the charts it is clear that the experimental diet has a much higher vitamin-C content than even the so-called good diet, and so results in a much higher daily urinary output. The value of a preliminary period during which the vitamin-C reserves of the body are filled up by the daily administration of large doses of vitamin C is shown by the higher levels of vitamin-C output of cases so treated.

Most rheumatoid arthritic cases are obviously unsaturated, and there would appear to be some factor other than the antecedent deficient diet causing this effect. Varying with the severity of the condition, there was much delay in obtaining both a rise and saturation—this difference is still greater when compared with the finding in the control cases who received similar doses of ascorbic acid after a preliminary period of hospital diet.

These results are in agreement with those of Greenberg, Rinehart, and Baker (1936) who found in a group of cases of active rheumatoid arthritis uniformly low ascorbic acid blood values, many of which cases were refractory to treatment with vitamin-C. The same authors and Harris (1937) have also shown that in acute rheumatic fever the vitamin-C content of blood and urine is low, and they too require large doses of vitamin C to bring these values to normal. Similar results are found in other infections (Harris 1936).

There was undoubtedly a very definite clinical improvement in all the patients on the high vitamin-C intake, but though these findings and those of other workers suggest very strongly that, though these rheumatic conditions are in some way related to a condition of vitamin-C unsaturation, it is impossible to state that the improvement is directly due to the increased vitamin-C intake. It may be found on further investigation that the low sodium-chloride output is of definite significance. It is certain that the low chloride intake is responsible for the loss of fluid round the joints; at the same time no consistent changes were present in the blood sodium chloride, the normal balance being kept by the loss of fluid. However, further work on the various salts of the blood are in progress.

CONCLUSION

1. Six cases on a high vitamin-C diet for rheumatoid arthritis have been investigated; one could not be kept strictly to the diet.

2. There was definite clinical improvement noticed in the other 5.

Our thanks are due to Dr. Annie Cunning who very generously financed the work, and to Miss Demery and Miss Cheek (Annie Cunning Scholars) who assisted in the investigation; also to Sister Mangan and Sister Brier who have supervised the patients in the wards.

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PREVALENCE OF SONNE ENTERITIS

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Several outbreaks of Sonne dysentery in institutions and similar communities have been recorded during the past few months. The disease is not confined to epidemic outbreaks in institutions, but is liable to occur endemically over widespread areas.

As was emphasised by A. F. Hurst and F. A. Knott in 1936,[1] the innocuous outcome and especially the short duration of this illness renders its occurrence very liable to pass unrecognised, and it is in general only when mass infection occurs that the expense of bacteriological investigation is undertaken and the diagnosis established. Hurst and Knott also pointed out that the condition was one of gastro-enteritis rather than of true dysentery, and with this our experience leads us to agree. In fact, the disease more closely resembles food poisoning, in the acuteness of its onset, the presence of vomiting in most cases, and the very transient nature of the illness. Although blood as well as mucus is usually present in the stools on the first or second day, this rapidly clears and there is nothing to suggest that permanent damage to the mucous membrane of the lower bowel occurs.

AN OUTBREAK IN A COLONY

In November, 1937, an explosive outbreak occurred among the male inmates of a colony for mental defectives in Wiltshire. One case occurred on the first day of the outbreak, three cases on the second day, twelve cases on the third day, and about thirty cases in all within five days among a total of about one hundred inmates in the infected wing. Experience shows that the incubation period of Sonne infections is very short (within a few hours), and there is little doubt that the infection was spread to some extent by personal contact. The symptoms were typical of such outbreaks, sudden onset of vomiting followed by severe diarrhœa with blood and mucus in the stools, severe prostration in some cases, and pyrexia in almost every case of 100°–103° F. Though blood and mucus were usual, cases occurred in which no more than a severe loose diarrhœa was present. Nearly all the cases showed recovery within three days, after which time the condition of the stools became normal.

Although recovery was so rapid, the condition of many of the patients during the acute stages gave rise to considerable alarm, and in one case the prostration was so severe as to give rise to the greatest anxiety and necessitate urgent action. Treatment in general was merely sedative, and gave excellent results. There did not appear to be any indication for the use of salines or of specific therapy. The ages of the persons affected varied from 16 to 40 years, and there did not appear to be any tendency to affect any particular age-group.

The outbreak was dealt with very promptly by Dr. D. H. H. Thomas, medical superintendent of the colony, and there is no doubt that the effective measures taken by him prevented a general spread of the infection throughout the colony. The sufferers and convalescents were rigidly segregated, and a search of the stools of the apparently healthy was made at once to detect carriers. Two carriers of *B. dysenteriæ* Sonne were identified among those who had not been affected with the disease, and it is significant that one of these was regularly employed in washing up dishes and plates at meal times. The segregation of these two persons along with the other sufferers brought an immediate end to the outbreak; only one case occurred subsequently, and this was directly traced to a malfeasance by a segregated ex-patient.

It is of interest to note that plates inoculated with fæces during the first twenty-four hours of the illness showed an almost pure growth of *B. dysenteriæ* Sonne, whereas those taken after the illness had lasted three days often showed only an occasional colony among the usual coliforms—in fact in some cases it was very difficult to prove the presence of *B. dysenteriæ* Sonne on the third day. *B. dysenteriæ* Sonne seems to be very rapidly crowded out by other intestinal habitants after the fæces have been passed, and in our experience plates should be inoculated at least within two hours of the stool being passed if reliable results are to be obtained. These factors suggest a further reason whereby cases may be missed, even when bacteriological investigation is utilised.

CASES OUTSIDE THE COLONY

While the usual investigations to determine the origin of the outbreak were being undertaken, it was decided to ascertain if the disease existed among the civilian populace outside the institution, since sporadic cases of diarrhœa were understood to be prevalent in this part of England at this time both in urban and in rural districts. The public health authority and the practitioners of Wiltshire are fortunate in enjoying a considerable measure of coöperation in the exercise of their respective functions, and practitioners were at once circularised by the county M.O.H., and their attention drawn to the desirability of investigating cases of sporadic diarrhœa. As a result of laboratory examination of fæcal specimens from about thirty such cases showing suspicious clinical symptoms, six persons suffering from Sonne infection were detected. These persons resided in different districts mainly in the south of the county and a neighbouring county, and were separated from one another by a distance of over fifty miles. Their ages reached from 9 months to 40 years, again demonstrating that the disease is by no means confined to infants and the young. In none of these districts had there been anything in the nature of an epidemic, though cases of diarrhœa were said to be fairly common. It thus seems probable that the condition of Sonne enteritis is much more widespread, especially in the late autumn, than is generally believed, and that it is an entity to be borne in mind by practitioners even in the absence of any explosive outbreak in their district.

TECHNIQUE OF DIAGNOSIS

It should be noted that the plates from the fæces of the thirty sporadic cases of diarrhœa mentioned above were examined also for colonies of *B. dysenteriæ* Flexner, with negative results. This rather confirms our experience over some years that sporadic cases of *B. dysenteriæ* Flexner infection are not common in this part of England and, though occasionally met with sporadically, this latter condition is much more commonly endemic in asylums and similar institutions, while *B. dysenteriæ* Sonne is more likely to be endemic among the general population. In such cases of

[1] *Lancet*, 1936, **2**, 1197.

B. dysenteriæ Flexner infection as we have seen, the specific organism is a habitant of the lower intestine for a considerably longer period than two days.

It was our experience during the outbreak described, as it has been in the past in other outbreaks of food poisoning, that specimens of actual fæces are preferable for bacteriological examination to rectal swabs. It is doubtless easier to collect specimens by the latter method, especially from children and the mentally deranged; but the difficulties of laboratory diagnosis are considerably added to thereby, as litmus lactose plates inoculated from rectal swabs are liable to show numerous pale colonies of late-lactose fermenting or *B. alkalescens* types which may closely resemble colonies of dysentery bacilli in appearance; the investigation and subsequent rejection of these colonies involves a great deal of wasted effort, and may easily lead to the dysentery colonies being missed. It would appear that organisms of the alkalescens type are common habitants of the lower rectum.

BACTERIOLOGICAL PITFALLS

In addition to the reasons already given that may cause cases of Sonne enteritis to be overlooked—i.e., transient illness, early disappearance of the specific organism from the stools, and the confusion caused by other non-specific pale colonies on lactose plate cultures—there is another factor which may greatly add to the difficulties of laboratory confirmation. This is the tendency displayed by some strains of *B. dysenteriæ* Sonne to extremely early production of acid from lactose. The standby of the clinical bacteriologist in the investigation of all acute bacterial infections of the intestine is the fortunate fact that the specific organisms, though of various types, lack in common the power to produce acid from lactose, at any rate for some days. Consequently, it is only the pale colonies which grow on litmus lactose plates which are considered for further investigation as potentially dangerous; the red and pink colonies are rejected straightaway as presumptive coliforms of no pathological significance. The strains of *B. dysenteriæ* Sonne with which we have been working during the past month all tend to produce acid from lactose very early; even on solid media (litmus-lactose bile salt-agar) the colonies cannot be relied upon to remain pale for more than twenty-four hours after inoculation of the plates, and by the end of forty-eight hours they are very definitely pink. It is therefore essential to choose the exact moment for picking off suspected colonies for examination; up to twenty-four hours all colonies are too small and undifferentiated to permit of reliable investigation, and if the selection is left much later, the *B. dysenteriæ* Sonne colonies will become pink and difficult to recognise from coliforms. The use of high titre "pure" specific antisera is absolutely essential for large-scale investigation of such outbreaks, and it is unfortunate that reliable and suitable sera are very difficult to obtain. The technique used by us follows that of Dr. W. M. Scott of the Ministry of Health laboratory. It consists of picking off with a straight needle a portion of a suspected colony, rubbing this up on a glass slide in (*a*) a single small loopful of specific *B. dysenteriæ* Sonne serum, (*b*) a single small loopful of Flexner serum. Should the suspected colony be *B. dysenteriæ* Sonne, fine granular clumping is visible, usually to the naked eye but always with a hand lens, within a few seconds in the case of the specific serum, with no clumping in the *B. dysenteriæ* Flexner serum. The remains of a positive colony is then picked off on to an agar slope for full subsequent cultural and serological confirmation. This simple procedure enables great numbers of plates and colonies to be examined rapidly, and gives very accurate results. The difficulty with the sera usually available is that they are either not sufficiently specific, and so liable to give false clumping, especially when concentrated serum is used; or so diluted that although quite adequate for macroscopic sedimentation tests in tubes where appreciable quantities of bacterial suspension are used, they fail to show sufficiently rapid naked-eye clumping when used directly on a slide with minute quantities of the suspected organism. We have investigated more than a dozen outbreaks of food poisoning, due to salmonellas, using this method, and have always obtained a solution within forty-eight hours (usually within twenty-four hours), which has subsequently in every case been confirmed as accurate by the more lengthy routine methods. We would again emphasise, however, that the essential of the rapid method is the possession of highly specific antisera which will give sharp differentiation between, for instance, a Dublin and a Newport strain salmonella. We again express our gratitude to Dr. W. M. Scott and to the Ministry of Health laboratory generally for familiarising us with the details and exact technique.

We have pleasure in expressing our appreciation to Dr. C. E. Tangye, M.O.H. for Wilts county, and to Dr. D. H. H. Thomas, both of whom afforded us every facility and assistance in the investigation.

Clinical and Laboratory Notes

RECIPROCAL RHYTHM

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The term reciprocal rhythm was first used by Drury (1924) in a case of nodal tachycardia to describe a mechanism of the heart in which a single impulse from the auriculo-ventricular node gives rise to two ventricular contractions. The impulse from the node passes down normally to the ventricle and causes it to contract. But the backward transmission to the auricle is so slow (retrograde heart-block) that after the auricular contraction the impulse is able to pass down again, and to reach the ventricle after that chamber has recovered from its refractory phase. So a second ventricular contraction results. Drury suggested that with the fast rate the conducting fibres might be partially refractory, and that some only might conduct the impulse back to the auricle while others might by that time have recovered sufficiently to conduct it forward to the ventricle.

A similar action had been noted under different titles by earlier writers in patients with nodal rhythm. The first example is a polygram by Wenckebach (1906). White (1915), who recorded the first electrocardiograms, suggested that the impulse-forming centre in the node might lie outside the ordinary conducting tract, and that the backward spread to the auricle and the forward spread down to the ventricle might not be along the same set of fibres. It must be remembered that all fibres in the auricle can conduct the impulse, and that there is no set path from the node to the ventricle.

CASE REPORT

A man aged 75 was seen with Dr. Mercer Jones on Sept. 26th, 1937.

In June he had had a heart attack with "missed beats" at his home for which he was kept in bed a week. Otherwise his cardiac history was negative. On the previous morning he had been found to be orthopnœic with a regular pulse-rate of 180. The blood pressure was 150–80 mm. Hg. He was given Nativelle digitalin granules (gr. 1/240) every four hours, and by evening the pulse-rate was 170 with occasional dropped beats.

The following morning the pulse-rate was 155, the rhythm being regular except for periods of intermission when beats were dropped. The apex impulse was outside the nipple, and mitral regurgitation was present. The lungs were congested. An electrocardiogram (Fig. 1) shows a tachycardia of doubtful origin, probably auricular flutter superimposed upon a left bundle-branch block. At noon on that day the pulse-rate was 120 and totally irregular. By the evening it was 80 and regular except for occasional extrasystoles. Digitalis was reduced to one tablet twice daily, and was stopped on Sept. 29th.

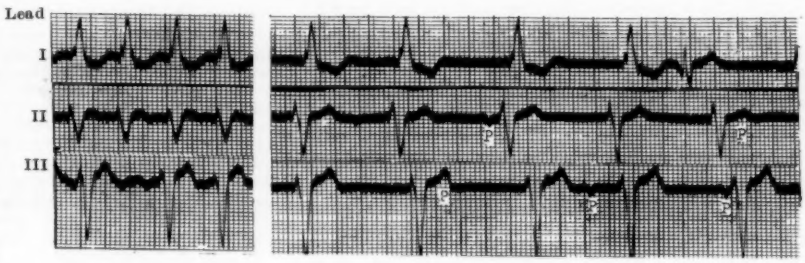

FIG. 1.—Sept. 26th: Auricular flutter and left bundle-branch block.

FIG. 2.—Oct. 4th: Left bundle-branch block, nodal rhythm with retrograde heart-block and reciprocal rhythm.

In order to confirm the diagnosis of auricular flutter, strongly suggested by the mode of termination of the paroxysm, a second electrocardiogram was taken on Oct. 4th (Fig. 2). This shows the anticipated bundle-branch block, but in addition the P waves are abnormal. A regularly recurring cycle of five beats runs through the record. It is continuous except in lead I where it is broken by a ventricular extrasystole. One cycle only need be described. The third complex in lead II is a beat of nodal rhythm with an inverted P preceding the Q R S by 0·12 sec. In the next complex no P wave is visible, being buried in the Q R S. In the last complex of that lead an inverted P occurs shortly before the end of the T wave. The Q–P interval in this beat is 0·28 sec. In the second complex of lead III the Q–P interval is also 0·28 sec. In the third complex an inverted P occurs after the end of the T wave, the Q–P interval being 0·48 sec. The next Q R S is a response to this P, the P–Q interval being 0·28 sec. The beat is slightly premature, arriving after 0·76 sec. in contrast to a constant rate of 0·92 sec. for the other beats in the lead. The cycle recommences at the next beat.

The record shows therefore nodal rhythm with a gradually increasing retrograde heart-block — a reversed Wenckebach period. When the Q–P interval reaches 0·48 sec. a reciprocal ventricular beat occurs.

COMMENTS

In most of the recorded examples in nodal rhythm the reciprocal rhythm has taken the form of bigeminy. The Q–P interval is consistently long and the P is always answered. A gradual increase in the retrograde heart-block with an initial inverted P preceding the Q R S is rare. The original polygram of Wenckebach was of this type. Blumgart and Gargill (1930), out of four cases showing reciprocal rhythm, included one (No. 4), also with a cycle of five beats. Reciprocal rhythm occurred when the Q–P interval reached 0·28 sec. Heart failure was present. Dock (1928) reported another, but in his case the upright P waves suggest dissociation with interference as a more likely explanation of the arrhythmia. In the case now recorded the patient had recovered from his attack of flutter and was in his usual health.

SUMMARY

A case is described of nodal rhythm with retrograde heart-block and reciprocal rhythm following a paroxysm of auricular flutter.

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A MODIFICATION IN THE TREATMENT OF EXTENSIVE VARICOSE VEINS

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R. Milnes Walker, M.S. Lond., F.R.C.S. Eng.
hon. surgeon to the hospital

The technique here described is a modification of the usual procedure of ligating the main vein for general or local varicosity of the long saphenous system, the object being to produce a thrombosis in the varicose veins (which have previously been made relatively empty) throughout their entire extent, and in continuity, by a single operation. The only special apparatus required is a needle 8–10 in. long which will fit a standard Record syringe. (The type used for splanchnic infiltration of local anæsthesia is convenient.) On the end of such a needle is fixed a copper ball ¼ in. in diameter through which the blunted needle passes.

Technique.—With the leg elevated, allowing a few minutes for blood to drain from the limb, the saphenous vein is exposed under local anæsthetic at the fossa ovalis and ligatured, leaving a second ligature just distal to the first; the vein is incised below the proximal ligature, whilst bleeding is controlled by tension on the distal one. The ball-ended needle is passed down the distal part of the vein to its full length, the rounded end allowing this to be done without fear of perforating the vessel wall. A 20 c.cm. syringe is attached to the needle and the vein is emptied as far as possible by aspiration, which procedure is further aided by massaging the limb in an upward direction. The ball-end prevents the wall of the vein being sucked against the lumen during this process. A second syringe containing 8 c.cm. of 5 per cent. sodium morrhuate is fixed to the needle and 4–5 c.cm., according to the size and extent of the vein, is injected under fair pressure, the remainder of the injection being

continued more gently whilst the needle is withdrawn. The distal ligature is tied and the vein severed. The measures taken to reduce the vascularity of the limb are maintained for a few minutes after the operation has been completed, during which time the limb is massaged in a downward direction.

The patients are allowed up on the third day and are back at work in a week's time. Very little post-operative pain has been noticed. In all cases the varicosity has been gross, affecting the whole or part of the long saphenous system, and in some where the varicosity has been as extensive the thrombosis has been continuous from the fossa ovalis to the medial malleolus. The immediate results, both curative and cosmetic, have been very good. Recently 14 patients were examined who had been treated by this method 8–10 months previously. Policemen, bus-conductors, and a blacksmith were amongst their number. In all but one there was no visible evidence of varicose veins; in that case a small lump could be seen which had previously been an enormous varix. None of them had had any further trouble, and all were well satisfied with the appearance of their limbs.

The advantages of this modified technique are:—

1. That the operation can be performed in a few minutes under local anæsthetic. There is no need for the further injections that so often become tedious to both patient and surgeon.

2. The thrombosis takes place with the minimum of blood in the veins, which causes a more lasting obliteration and a much better æsthetic result.

3. The venous channels are thrombosed in continuity so that unobliterated portions of the vein do not remain communicating with the deep veins of the limb, which is so often the case where multiple injections have been used, and is a potent cause of recurrence.

4. The saphenous vein having been ligatured before the injection is made, there is no danger of the separation of any clot of appreciable size and of consequent embolism.

The type of case suitable for this line of treatment is the one in which there is generalised or localised varicosity of the long saphenous system, provided that the valves are incompetent and the deep veins patent as shown by the tests of Trendelenburg and Perthes. We may add that associated ulcers heal in a remarkably short time.

MEDICAL SOCIETIES

NORTH OF ENGLAND OBSTETRICAL AND GYNÆCOLOGICAL SOCIETY

A MEETING of this society was held in Liverpool on Nov. 26th, 1937, with Dr. J. W. BRIDE, the president, in the chair.

Sulphanilamide in Obstetrics

Mr. BRYAN WILLIAMS (Liverpool) read a note supplementing his paper on the use of Prontosil in obstetrics read at Newcastle-upon-Tyne last June.[1] Unless there was vomiting that prevented the swallowing of large numbers of tablets he now gave sulphanilamide by mouth rather than by injection. Altogether, he said, he had treated 84 cases of puerperal sepsis. In 44 the hæmolytic streptococcus was not found, and he had no evidence that prontosil was of value in such cases. In 40 cases a hæmolytic streptococcus was isolated. In 19 of these, where the infection was localised to the perineum, vagina, and uterus, there were no deaths. In 10 cases where the infection involved the pelvic cellular tissue, peritoneum, veins, Fallopian tubes, or ovaries there were again no deaths. In the remaining 11 cases either septicæmia or peritonitis or both were present, and 6 were fatal; but in 5 of these 6 either the patient was extremely ill on admission or doses were given that were now known to be inadequate. No patient had died since his last report. He had also used the drug prophylactically, and had found no hæmolytic streptococci in 104 cases where it was given, whereas a hæmolytic streptococcus was found in 8 of 73 controls. The more important of the toxic effects of the drug were jaundice, acute hæmolytic anæmia, agranulocytic angina, skin eruptions, and neuritis. Cyanosis had been prominent, but in the more serious cases he had continued treatment in spite of it. It was clear that there had been a fall in the fatality of hæmolytic streptococcal infections since the introduction of these drugs, but at the same time there was evidence of a fall in the virulence of such infections in recent years. Nevertheless there was no doubt that sulphanilamide, in adequate doses, substantially lessened signs and symptoms caused by the hæmolytic streptococcus, especially when the patient was seriously ill with peritonitis or septicæmia. Indeed if this improvement did not occur, it could be assumed that a hæmolytic streptococcus was not responsible for the illness. Though there was no justification for the indiscriminate use of the drug, it was in his opinion valuable as a prophylactic. It should not be used as a routine.

Dr. S. B. HERD said that he had sometimes found that cases which did not respond at first to prontosil responded after a blood transfusion.—Prof. JOHN CHISHOLM (Sheffield) said that the drugs had been used a great deal at the Jessop Hospital, and the results had been comparable with those of Mr. Williams.—Mr. A. GEMMELL quoted a case in which there was a pyrexia apparently due to prontosil.—Mr. C. H. WALSH spoke of the possibility of idiosyncrasy.—Mr. J. W. BURNS had seen a case in which cyanosis persisted for a month.—Mr. T. N. A. JEFFCOATE thought that cyanosis depended on the patient and not on the dosage. He had seen pyrexia and jaundice following the use of sulphanilamide.

In reply Mr. WILLIAMS said blood transfusion had been carried out in many cases with evident improvement. The dose of sulphanilamide he gave was four half-gramme tablets 4-hourly; after a few days this was reduced by a third.

Toxic Hepatitis

Mr. C. J. K. HAMILTON (Liverpool) reported a case of "acute yellow atrophy."

The patient, aged 32, who had had a normal confinement in 1935, was admitted to hospital on August 10th, 1937, at the 36th week of pregnancy. A week previously she had had attacks of orthopnœa. On admission her temperature was normal but the pulse-rate was 120. She was drowsy and slightly jaundiced. The height of the fundus corresponded with 36 weeks, and there was some diminution of dullness over the liver.

A classical Cæsarean section was performed under local anæsthesia, and a living child (6 lb. 10 oz.) delivered. Intravenous 10 per cent. glucose saline was given by the drip method with 10 units of insulin in each pint. Abdominal distension increased and the jaundice became deeper. On the second day after delivery the jaundice was very deep and the patient nearly comatose. Liver dullness was absent, the temperature had now risen to

[1] See *Lancet*, 1937, **2**, 83.

104° F., and the patient had two rigors. She died on the third day after delivery.

The liver weighed 40 oz. and was yellowish. Section showed red areas of necrosis surrounded by areas of fatty degeneration. Microscopically there was necrosis and fatty infiltration of the liver cells in the central areas.

The infant was slightly jaundiced the day after operation. The jaundice increased on the second day, and paternal blood, and later serum, was given for the ensuing four days, during which the jaundice gradually cleared. Seen at about 11 weeks old, it was apparently well in every way.

Mr. Hamilton thought that the jaundice of the child was interesting. Dudley in 1901 had reported a case of normal delivery in which the child was born jaundiced.

Dr. N. B. CAPON spoke of the relationship between icterus gravis, anæmia gravis, and congenital œdema of the fœtus; cases seen in this country seemed to show erythroblastosis much less frequently than those recorded in America. Early treatment by intramuscular injection of human serum was of the greatest importance, and maternity hospitals should keep a supply of serum available for use in emergency. As soon as the child was out of danger, and the jaundice was subsiding, blood transfusions were of value to overcome the consequent anæmia.

Mr. C. H. WALSH reported a case seen at the same time as Mr. Hamilton's.

The patient was admitted at the 24th week of pregnancy, with right-sided pain and vomiting for the previous five days. There was a slight jaundice but her condition was fair. At a previous pregnancy she had had jaundice 7 weeks after delivery, but this had cleared up in a few days. The urine contained pus and *B. coli*. She was treated with calcium gluconate and glucose, and the membranes were ruptured to induce abortion. She was delivered on the following day and died two days later. Autopsy showed typical liver necrosis.

Mr. Walsh suggested that this was a case of toxæmia in which the toxin affected the liver primarily, with no damage to the kidneys. Toxic jaundice might be a terminal condition in some cases of severe toxic vomiting. Possibly a specific toxin was elaborated, which had a selective destructive effect on the liver cells in toxic jaundice.

Endometriosis

Prof. A. M. CLAYE (Leeds) reported the case of a woman of 30, who had complained of dysmenorrhœa and dyspareunia for several years, and had been married nine years without pregnancy. An indefinite mass was felt behind the uterus, and at operation in March, 1936, a chocolate cyst of the left ovary was found, which burst and was removed. The right ovary was enlarged and showed multiple cysts. Wedge enucleation was performed, and after the operation dyspareunia was relieved, and dysmenorrhœa much less. When seen again the day before the meeting she was 32 weeks pregnant. Prof. Claye's hospital records showed only two other examples of pregnancy after a conservative operation for endometriosis. He had met with endometriosis at Cæsarean section on three occasions. Bourne and Williams said that nearly half of the patients with endometriosis were sterile. This might be due to two kinds of ovarian dysfunction associated together. In some cases the adhesions in the tubes and in others the dyspareunia militated against conception. Where the disease was not very advanced, efforts should be made to leave some ovarian tissue to preserve the possibility of pregnancy. His impression was that the age-period of the disease was placed too highly by most authorities.

The PRESIDENT and Prof. MILES PHILLIPS (Sheffield) had each seen a case of endometriosis in which pregnancy had supervened. The latter thought that artificial prevention of pregnancy in early marriage might possibly be the cause of endometriosis.—Mr. ST. GEORGE WILSON was of opinion that many cases of chocolate cyst could not be demonstrated as endometriomata, and it was in these cases that pregnancy occurred.—Prof. LEYLAND ROBINSON, Mr. JEFFCOATE, and Mr. GEMMELL had all seen cases similar to Prof. Claye's.

In reply Prof. CLAYE said he thought the incidence of sterility was exaggerated.

Breech Presentation

Mr. ST. GEORGE WILSON showed a cinematograph film entitled a Method of Delivering the Trunk and Arms in Breech Presentation. He said that the *Journal of Obstetrics and Gynæcology of the British Empire* for August contained an article by Løvset of Bergen on shoulder delivery in breech presentation. The theory was that at a given moment the posterior shoulder was lower than the anterior shoulder owing to the inclination of the pelvis. When the posterior shoulder had got below the promontory of the sacrum, the body of the fœtus was rotated through 180°, and the posterior shoulder then came behind the symphysis and the arm appeared below the pubic arch. The arm was easily freed and the trunk rotated back again through 180°, when the remaining arm appeared under the pubic arch. These facts were demonstrated in Mr. St. George Wilson's film and he claimed (1) that the method was easy; (2) that displacement of the arms was an advantage rather than a hindrance; and (3) that the risk of laceration and sepsis was less as the hand was not introduced into the vagina.

Mr. BURNS thought the method had nothing to recommend it. There was a risk of breaking the fœtal neck by twisting.—Prof. PHILLIPS, Mr. GEMMELL, and Mr. GLYN DAVIES however all regarded the method as excellent and had used it for years. Prof. Phillips pointed out that it was originally adopted by Potter of Buffalo.

THE LANCET 100 YEARS AGO

December 30th, 1837, p. 495.

From a letter from a student of University College.

SIR:—The just and fearless manner in which THE LANCET has ever been devoted to the advancement of medical education, induce me to hope that you will continue to give your attention to the Medical School of *University College*. The nomination to the chairs in this School is entirely in the hands of the Council, and it gives me great pleasure to bear evidence to the fact that the majority of them are filled quite to the satisfaction of the students. . . . In the department of chemistry . . . the present Professor . . . although well-informed in his subject, and thus far highly qualified for the appointment, . . . is deficient in one essential at least, which is, perhaps, more necessary than even profound chemical research itself. In France, and throughout Germany, a clear and distinct enunciation, and a capacity for imparting information, are considered to be *indispensable* qualifications in a professor. In these points Professor Graham is exceedingly defective. He is by birth, and early association, so inveterately Scotch in his accent, as to be often unintelligible to his English auditors.

I endeavour to get as near as possible to him at lecture, yet I, day after day, leave the theatre uninformed on matters on which he discourses, as I am never able to follow him through a sentence of any length. . . .

REVIEWS AND NOTICES OF BOOKS

Developmental Abnormalities of the Eye

By IDA MANN, D.Sc., M.B., B.S. Lond., F.R.C.S. Eng., Surgeon, Royal London Ophthalmic (Moorfields) Hospital; Ophthalmic Surgeon, Royal Free Hospital. Published for the *British Journal of Ophthalmology*. Cambridge: at the University Press. 1937. Pp. 444. 50*s*.

THIS work is an interesting sequel to a book on the development of the human eye written some years ago by the same author. In many respects the two books are complementary. This one is a monograph which deals with the application of embryology to the elucidation of certain clinical problems. Miss Mann refers abnormalities of the eyes to three main stages or periods of development: the germinal, the organogenetic, and the fœtal. She states that the time of appearance of a defect is no indication of whether it is or is not genic in origin, for its manifestation may be long postponed. In one group of congenital abnormalities the departure from the normal is that of disposition of parts only, no genic or chromosomal alteration having occurred; such defects are due to environmental causes (chemical, osmotic, or toxic), but it is impossible to determine their nature since the result of interference with growth during the organogenetic period is specific for the time at which the noxious agent acts. "Thus disorders of growth arising during the organogenetic period will produce, if early, deformities rather than arrests, if a little later, arrests associated with aberrant growth (deformity), and later still simple arrests or retardation; the type of abnormality is thus determined by the time of action and not by the nature of the agent. Diseases of the fœtus resemble those of the adult the more closely the later they occur." In discussing the ætiology of oxycephaly Miss Mann surveys theories advanced by other writers on this subject and presents a suggestive conception of her own. She maintains on good evidence that oxycephaly is a true developmental anomaly of germinal origin, a dysgenic condition manifested by a localised arrest in the development of the post-optic visceral mesoderm (maxillary process) which may fail to respond to the widening of the brain and may remain in a retarded atavistic condition, the process beginning during the organogenetic period.

Perhaps the best part of this admirable work are Chapters VI and VII on the iris. The text of this section contains much original work by the author, and she is able to draw material from her wide range of knowledge about comparative anatomy. The omissions are few; one that we noticed is in the description of Bourneville's syndrome, where there is no mention of adenoma sebaceum, an important clinical feature in this disease. Many of the illustrations drawn and painted by the author are reproduced in colour and show a wealth of informing detail. The three coloured illustrations of cerebromacular degeneration are particularly interesting, for they show the disease in its early stages. Unfortunately the pictures of the histological features of glioma (neuroblastoma) retinæ are poorly produced.

This is not an exhaustive survey of all the known deformities of the eye, and the author is well aware that her keen interest in certain problems has affected its balance. She admits also that further advances in embryological knowledge or new methods of examination may disturb many of the theoretical conceptions that she has elaborated; but these hypotheses enliven the work and its individual flavour is enhanced by the crisp enunciation of the opinions she holds and her grounds for holding them. Miss Mann is well qualified both as an embryologist and a clinical ophthalmologist to speak with authority on such matters, and the enthusiasm with which she has attempted to correlate developmental anomalies of different types with the various stages and processes of development in which they originated makes this book of exceptional value both to anatomists and to clinicians.

Diseases of the Blood and Atlas of Hæmatology

By ROY R. KRACKE, M.D., Professor of Bacteriology, Pathology, and Laboratory Diagnosis, Emory University School of Medicine; and HORTENSE ELTON GARVER, Instructor in Laboratory Diagnosis, Emory University School of Medicine. London: J. B. Lippincott Company. 1937. Pp. 532. 65*s*.

THIS atlas of blood disease is a portentous volume, of which no less than 24 pages are devoted to definitions of hæmatological terms. The arrangement of the letterpress is difficult to follow. The anæmias are discussed in a haphazard way—for instance, the different types of hæmolytic anæmia are described in widely separated chapters. In the table classifying the leukæmias, megakaryocytic leukæmia is included as a distinct type ranking with myelogenous leukæmia in importance, but is never mentioned or described again. The brief chapter on the bone-marrow by R. P. Custer is interesting, and in the account of the blood picture in normal laboratory animals much helpful information is collected from scattered sources. The discussion on hæmatological technique is comprehensive. There are 44 full-page colour plates in this atlas.

Blood-cells unfortunately are pretty things—their charm is a snare and a delusion. Until Whipple, Minot, and Castle, puritanical experimentalists, ignored the fine points of morphology, hæmatology was largely an affair of picture books. Men quarrelled over the nomenclature and genealogy of blood-cells but both the ætiology and the therapeutics of blood diseases remained obscure. Hæmatology to-day is alive because it has become an experimental science. The increasing number of elaborate hæmatological atlases is therefore to be deplored because they inevitably lay emphasis on the less important aspects of the blood dyscrasias; and it is unlikely that students, clinicians, or research workers will learn much of value from this one.

Story of a Great Hospital: The Royal Infirmary of Edinburgh, 1729–1929

By A. LOGAN TURNER, M.D., LL.D., Hon. F.R.C.P. Edin., F.R.C.S.E., Consulting Surgeon, Ear, Nose, and Throat Department, Royal Infirmary, Edinburgh. London and Edinburgh: Oliver and Boyd. 1937. Pp. 406. 10*s*.

THIS book has much more than local interest, for Dr. Logan Turner traces clearly the development of the voluntary hospital in Britain, showing how the Royal hospitals in London helped to bridge the gap between the monastic hospital and the voluntary subscription movement of the eighteenth century. But its chief concern is of course with Edinburgh. The

"Father" of the Edinburgh medical school was John Monro, who in common with many Scottish students of his period derived inspiration from his studies at Leiden. His son, Alexander Monro, along with other founders of the school, worked with Boerhaave, and Dr. Turner points out that the emphasis which Boerhaave laid on clinical teaching has remained a lasting feature of the Edinburgh tradition.

The gradual development of the Royal Infirmary provides a story of absorbing interest, and the wisdom and courage of its managers in meeting the ever-changing and increasing demands made on the institution is impressive. The provision for wounded soldiers after the '45 rising; the beginnings of hospital midwifery; the provision of a public bath-house in 1756; the threatened expulsion of James Syme from the staff; the reason why John and Charles Bell left Edinburgh; the power of John Rutherford (Walter Scott's grandfather) in the school; the influence of the Edinburgh methods of teaching on the developing American colleges; and the personalities of Simpson, Syme, and Lister, are among the matters discussed. The later pages are concerned with the astonishing developments of the last fifty years, and at the end of the book are tables illustrating the growth of the hospital, and lists of former and present members of the staff. The illustrations include portraits and prints of old Edinburgh buildings. The book is beautifully produced.

Trauma and Disease

Edited by LEOPOLD BRAHDY, B.S., M.D., Physician in Charge of Industrial Diseases and Accidents in the Office of the Corporation Counsel of the City of New York; and SAMUEL KAHN, B.S., M.D., Medical Examiner in the Bureau of Workmen's Compensation of the Department of Labor, State of New York. London: Henry Kimpton. 1937. Pp. 613. 35s.

THE temptation to trace a causal relationship between the occurrence of an accident and the subsequent onset of disease, however much later, is a very old one. But the doctrine of proximate cause as exemplified by the relation of trauma to disease has become in these days a much more fertile source of litigation than it used to be. The counsel attempts to earn the fee marked on his brief by drawing lurid pictures of the osteo-arthritis that is certain to follow the most trivial mishap to bone or joint; every strained back that reaches the law courts is an incipient spondylitis deformans; and traumatic epilepsy and insanity are almost equally certain to follow a blow on the head. An authorised treatise on trauma and disease has long been needed, and this one was well worth waiting for. It would of course be impossible to survey the field in 600 pages, but the principles here laid down are sufficient to guide the examiner in most problems likely to confront him. The authors have bravely tackled the subject from its more difficult angle. It is easy to argue from trauma to disease; it is not so easy to start with disease and then trace back "bridging symptoms" to a specific trauma. But this is where one has to start in practice, and the book is made much more valuable by this approach.

Three groups of diseases are here considered in relation to trauma:—

1. Those that are practically always the direct result of trauma such as fractures. This group is not discussed in detail in the book.

2. Those which are never the result of a single trauma. Excluding diseases in which a traumatic origin is out of the question (e.g., measles) there are others, such as colloid goitre or Köhler's disease of the metatarsals, in which there might be a doubt and the evidence must then be carefully gathered and weighed. If as the result of such an investigation the conclusion has been reached that trauma is never an ætiological factor, and the disease is placed in the second group, the question of causal relationship between the trauma and any specific case of this disease need not arise.

3. Those which usually develop without trauma but in which trauma may be a causative factor—angina pectoris, kidney abscess, exophthalmic goitre, and so forth. The relation of trauma and disease in this particular group must be considered separately in each case.

Experimental evidence can sometimes be brought to bear on the connexion between trauma and some specific disease; this is of course much more valuable when it is positive, for negative results can hardly disprove such a connexion. Statistics are obviously most useful but they lead to fallacious conclusions unless interpreted by an expert. For example, in a series of 431 cases of glioma reported by Dr. Kernohan a history of injury was given in 13·4 per cent. In a control group of 200 presumably normal persons there was a history of injury to the head in 35·5 per cent. Few would defend a conclusion from such figures that injury to the head decreases the likelihood of the occurrence of cerebral tumour; but arguments almost as absurd have been solemnly enunciated and even gained currency. The sort of information in this book that will be found really useful is that which records the later history, for example, of a large group of cases of injury to the peripheral nerves during the late war. Not a single instance of paralysis agitans, multiple sclerosis, or progressive muscular atrophy (or for that matter any complicating neurological disease) was found among them. The writer of this section adds: "With the exception of residuals of direct trauma to the brain, spinal cord, or peripheral nerves, I do not recall one instance of disease of the nervous system that I can attribute solely to trauma."

Amongst the 23 contributors there are some rather disconcerting differences of opinion. Discussing trauma and disease of the gastro-intestinal tract B. B. Crohn quotes the four postulates of Liniger and Molineus for traumatic ulcer of the stomach as a fair beacon or guide to those considering all questionable cases. The postulates are: (1) that no known disease existed before trauma; (2) that the trauma be sufficiently severe to act as a causative factor; (3) that the trauma be immediately followed by active symptoms of the disease; (4) that the condition appearing after the trauma present the clinical symptoms of the disease alleged to have been produced and be otherwise identical with it. Hugh Cabot on the other hand, writing on tuberculous infection of the kidney, says: "It may, I think, properly be held that any injury of sufficient severity to lower the resistance of the tissues of the kidneys may operate to produce the disease. Obviously such an injury need not be severe, need not be disabling and need not produce any obvious clinical symptoms such as hematuria, ecchymosis or visible bruising of the tissues."

Such conflicting theories cannot be put down to the fact that each refers to a different system of the body. The difference is more fundamental and shows that the subject of trauma and disease still bristles with difficulties. But most of the conclusions

reached in this book are based upon common sense and logic. It is agreed that an opinion cannot always be categorical. This is not perhaps a book for the inexperienced witness, but the expert who realises his heavy responsibility in influencing the courts for or against the compensation of some victim of circumstance will find much support in its pages.

Thorpe's Dictionary of Applied Chemistry

Fourth edition. Volume I (A–Bi). By JOCELYN FIELD THORPE, C.B.E., F.R.S., F.I.C., Professor of Organic Chemistry and Director of Organic Laboratories, Imperial College of Science and Technology; and M. A. WHITELEY, D.Sc., F.I.C., lately Assistant Professor of Organic Chemistry at the College. London: Longmans Green and Co. 1937. Pp. 703. 63*s*.

THE final volume of the last edition of "Thorpe's Dictionary" having appeared 10 years ago, the editors decided that the great progress since made in applied chemistry demands the issue of a completely new edition. They intend to publish a volume a year, and in order to allow for future advances they have adopted a mechanism whereby each volume will be used as a means of bringing a previous one up to date. The fact that there are nearly eighty contributors to this volume demonstrates their realisation "that the day of the universalist is past and that the work in any special field must be described by an expert thoroughly acquainted with his subject."

The dictionary contains a vast amount of pure chemical information; and pharmacology and biochemistry, as two of the most important forms of applied chemistry, are given a good deal of the available space. Of particular interest to those concerned with the medical sciences are the articles on alkaloids, amino-acids, vitamins B and C, bile, bile pigments, autolysis, anæsthetics, and barbituric acid. The editors aim to provide articles so written as to be understood by the ordinary cultured reader, and indeed some of the articles—e.g., on aerated waters, on alcohol, and on accelerated ageing (of protective coatings)—make fascinating reading for those quite ignorant of their subject. Not the least pleasant feature of the volume is the binding, which is coloured by the new dyestuff, Monastral Fast Blue.

Malaria and Ankylostomiasis in the Pregnant Woman

By G. A. W. WICKRAMASURIYA, F.R.C.P., F.R.C.S. Edin., M.C.O.G., Visiting Obstetrician, De Soysa Lying-in Home, Colombo; Tutor in Clinical Midwifery, Ceylon Medical College. London: Humphrey Milford, Oxford University Press. 1937. Pp. 179. 10*s*. 6*d*.

THE work is based upon the essay that gained the Katherine Bishop Harman prize for 1936. It contains a mass of detailed material collected upon a subject which, in view of its importance, has been singularly neglected. In Ceylon the bulk of the population suffers from malaria and ankylostomiasis, and it is not unreasonable to attribute the high maternal and infantile mortality in the island (20·6 and 173 per thousand respectively for 1934) to the ill health of the prospective mothers. On the whole Dr. Wickramasuriya holds ankylostomiasis to be a greater menace than malaria owing to its wider prevalence and its more insidious course; it may and often does coexist with malaria. That both ankylostomiasis and malaria render women more liable to the toxæmias of pregnancy, such as eclampsia and pre-eclampsia, is a fact that has hitherto escaped adequate recognition.

The author of this book has brought forward convincing evidence of the passage of the malaria parasite to the fœtus, thus placing the conception of congenital malaria on a secure basis. He regards quinine as indispensable in the treatment of gravid women, and quino-plasmoquine and atebrin as adjuncts that may be necessary. Far from being an oxytocic drug, quinine in effective therapeutic doses definitely prevents the premature interruption of pregnancy and intra-uterine death of the fœtus. Preliminary blood transfusion often averts the dangerous collapse that may follow delivery in women suffering from malaria. Abortion, miscarriage, premature and stillbirths are common among women suffering from hookworm disease and some 90 per cent. of the patients exhibit albuminuria and anasarca in the second half of pregnancy.

Pediatric Urology

By MEREDITH F. CAMPBELL, M.S., M.D., F.A.C.S., Professor of Urology, New York University College of Medicine; and JOHN D. LYTTLE, A.B., M.D., Assistant Professor of Diseases of Children, College of Physicians and Surgeons, Columbia University. London: Macmillan and Co. 1937. Pp. 576 and 540. Two volumes. 63*s*.

THIS book, we learn, is the product of ten years' work. It consists of two volumes of which the first is divided into four sections, one dealing with methods of examination and diagnosis, the second with obstructive uropathy, and the third with anatomy, physiology, embryology, and anomalies of the urogenital tract, and the last with urinary infections. The second volume consists of eight sections, the first being devoted to Bright's disease. This term is used to cover acute and chronic glomerulonephritis "hæmorrhagic Bright's disease," nephrosis, chronic pyelonephritis, and renal rickets. Bright originally described a kidney lesion in a sailor; renal rickets, or nephrosis, had no place in his original description, and it is questionable whether they should have a place in the section under this title. Some would say that in a book that will probably be read more by surgeons than by physicians, the sixty odd pages devoted to Bright's disease are in any case redundant, but since pædiatrics is a common field there is much to be said for its inclusion. The succeeding chapters are devoted to genital diseases, urogenital injuries, calculus, tumours, neuromuscular disease, enuresis, uro-surgery, and a bibliography of M. F. Campbell's publications. This work, which is a cross-cut of medicine and surgery, is very readable and the extensive quotations from clinical records kept by the authors add to its practical interest. Great stress has been laid upon congenital anomalies of the urogenital tract, which appear to be less prevalent in this country than in America. Doubtless, however, if all children attending British hospitals with any urinary symptoms whatsoever—i.e., enuresis—were subjected to a complete urinary investigation, including cystography and pyelography, the number of congenital anomalies recorded would be higher; those giving no recognisable symptoms now remain undetected. This book is profusely illustrated with excellent photographs, drawings, and reproductions of radiograms, and its index is well compiled. All interested in the dysfunction of the urogenital tract in children should have it at hand.

THE LANCET

LONDON: SATURDAY, JANUARY 1, 1938

ANTISERA IN RELATION TO CANCER

THE brilliant results achieved in the treatment of a few bacterial diseases by antisera have led to many attempts to apply the same principles towards the cure of malignant growths. The assumption underlying such attempts is that the conditions are analogous and that it might be possible to develop a tumour immunity corresponding to active and passive bacterial immunity. Analogies may be helpful or they may be dangerous; and it is pertinent to inquire into the basis of this one.

The foundation of our knowledge of bacterial immunity was the observation that when living creatures contract bacterial diseases they often recover from them and are thereafter resistant to subsequent infection from the same cause. They develop, as we say, active immunity. By adapting Nature's ways this resistant state may be transferred passively from one animal to another through the medium of antisera. Resistance of this kind has not been observed in malignant growths. That spontaneous recovery from cancer does occur is well attested in the records collected by ROHDENBURG [1]; the phenomenon is, however, so rare that it can hardly be likened to recovery from bacterial infections. Further, the fact that multiple primary tumours are not uncommon shows that one tumour does not give protection against another. There is one sense in which the phenomenon of resistance has been observed. If a transplanted mouse or rat tumour retrogresses, if a tumour graft fails to take, or if embryo or other homologous normal tissue is first injected, further attempts at tumour grafting in that animal will fail. But, as will be explained below, from our knowledge of human cancer there is little evidence in favour of such a condition as tumour immunity in the bacterial sense, and the very use of such a term is misleading. The inadequacy of the methods of treating cancer in current use is, in the minds of many, sufficient justification for exploring every likely and unlikely approach to better ones. As far as serum therapy is concerned, we have watched with interest over many years the attempts made by Dr. THOMAS LUMSDEN, working for the British Empire Cancer Campaign, to prove that it is possible to obtain an antiserum specifically lethal to cancer cells. Dr. LUMSDEN'S method of attacking the problem consists in taking rats which are resistant to grafts of Jensen's rat sarcoma—the resistance may be natural or may be a consequence of the retrogression of a tumour—and injecting into them, usually intraperitoneally, emulsions of Jensen's rat sarcoma. After five or six of such immunising injections the sera of the rats are shown to possess the property of killing Jensen rat sarcoma cells. The acquisition of this power appeared at first sight to be a distinctly hopeful phenomenon, but the hope abates when it is found [2 3] that the same serum kills normal spleen cells. With the further demonstration [3] that rats which are injected with *normal* rat spleen develop a substance in the blood-serum lethal both to spleen cells and to the malignant cells of the Jensen rat sarcoma, the evidence is complete that the antisera which have been called "anti-cancer sera" are not specific for cancer cells.

The subject of serum therapy in cancer is discussed in the thirty-fifth annual report of the Imperial Cancer Research Fund which we noticed briefly last week. One of the fundamental points brought out is that grafted tumours in mammals must be regarded as grafts and not as new tumours. The Jensen rat sarcoma cells growing in rats to-day are, according to all the evidence at present available, lineal descendants of the cells of a sarcoma which arose spontaneously in a rat in Copenhagen 30 years ago. Their genetic constitution is the same as, or possibly slightly different from, the constitution of the cells of the rat in which the tumour arose. They are genetically different, doubtless in varying degrees, from the normal rats into which they are now grafted in the process of transplantation. They are in fact foreign cells and the immunological responses are to their "foreignness." When bacteria are injected into test animals it is the bacterial protein foreign to the animals that evokes the antibody response. The morphology of the bacterium is not responsible for the reaction. If the protein is the same, wholly or in part, in organisms differing widely in morphology and nature, a common antibody will be evoked, as is the case with the rickettsia of typhus and *B. proteus* X19. The same is true of injected cells; serum antibodies which the cells provoke appear in response to the "foreignness" of the contained protein, and there is no other kind of discrimination on the part of the animal receiving the injection. All is overshadowed by the extremely sensitive protein response; malignancy or non-malignancy does not enter into the question. In view of this fundamental obstacle to the production of a discriminating antiserum it seems likely that further research along these lines will be abandoned as unprofitable.

ÆTIOLOGY OF RENAL CALCULI

THE urine normally contains certain relatively insoluble substances—of which uric acid, calcium oxalate, and the earthy phosphates are the most important—in far greater concentration than can be accounted for by simple solution. This apparent supersaturation is maintained either because these salts are present in the urine as more complex compounds of greater solubility, or because of the adsorptive action of colloids. Whichever explanation

[1] Rohdenburg, G. L., *J. Cancer Research*, 1918, **3**, 193.

[2] Lumsden, T., *Amer. J. Cancer*, 1937, **31**, 430.
[3] Phelps, H. J., *Ibid*, p. 441.

is correct, a very small alteration in the physico-chemical state of the urine suffices to cause deposition of its solids.

Deposition is favoured by any factor that increases concentration, such as diminished secretion in hot weather or in general infections, and excessive ingestion of calcium salts or their excessive excretion (e.g., in hyperparathyroidism). Changes in the hydrogen-ion concentration may also cause deposition, as may alterations in surface tension such as take place around a foreign body or an ulcerated patch of epithelium. Many people, however, pass crystals in their urine, and only a few have calculi. If calculi are to form, crystals must not only be deposited but also retained. Any cause of stasis will encourage retention, whether an actual obstruction or mere stagnation. Stones often develop in the course of treatment of compound fractures, of tuberculous disease of bones and joints, and of injuries to the spine. They may be due to an increased excretion of calcium in these conditions, but prolonged recumbency seems to be the main factor, and frequent changes of position with adequate intake of fluids overcomes the tendency. Crystals form more readily if there is a nucleus for them to form on, and in the kidney this may be provided by a clump of bacteria or a patch of desquamating epithelium. RANDALL, EIMAN, and LEBERMAN [1] report that a deposit of calcium salts in the form of a plaque in the basement membrane of the collecting tubules and in the intertubular connective tissue is a common finding, occurring in 23 per cent. of 609 general autopsies they have performed. They show that, although these plaques are harmless as long as they remain intrapapillary, they are liable, when they occur near the surface, to lose their epithelial covering and form a nidus in which urinary salts may deposit themselves. They were able to demonstrate such a plaque in almost all of 41 examples of renal calculi. How far infection plays a primary part in stone-formation is still disputed. Bacteria are often found to form the nucleus of infected calculi, and there can be no doubt that the urea-splitting bacteria, such as *Bacillus proteus* and *Staphylococcus albus*, aid in the precipitation of phosphates. It seems to be definitely established, however, that stones can form apart from any infection, though infection may evidently hasten their growth.

For some years it has been known that gross deficiency of vitamin A will produce renal calculi in animals, and some observers believe that such stones form more easily when vitamin D is present in excess.[2] Absence of vitamin A causes hyperplasia and keratinisation of the renal and vesicular epithelium, and may produce very much the conditions that RANDALL and his colleagues have shown to precede calculus-formation in human beings. The tests for vitamin-A deficiency by means of dark-adaptation now make it possible to ascertain whether such deficiency can be demonstrated in patients with calculi, and following this line EZICKSON and FELDMAN [3] have studied the vision of 25 adults who either had calculi or were known to have had them recently. They found that 24 had "pathological" dark-adaptation, varying from mild to severe, and they treated them accordingly with a vitamin-A concentrate providing 13,000–52,000 "units" daily for six to nine months. Of this group 15 returned for re-examination, of whom 11 had carried out their treatment regularly and 4 irregularly, and it is recorded that 14 of the 15 still showed definitely pathological dark-adaptation while the other was still not normal. Other workers who have investigated night-blindness (e.g., MAITRA and HARRIS [4]) have observed an immediate and rapid response to vitamin A in most cases and if patients with calculus show no such response they appear to be in a class apart. The question arises whether their deficiency symptoms are due, not to insufficiency of the vitamin in the diet—though such a deficiency is sometimes demonstrable—but rather to an inability to absorb or utilise it. It must, on the other hand, be remembered that vitamin-A deficiency is not the sole cause of night-blindness, though the simultaneous presence of defective dark-adaptation and urolithiasis in this group of cases is highly suggestive.

[1] Randall, A., Eiman, J. E., and Leberman, P. R., *J. Amer. med. Ass.* Nov. 20th, 1937, p. 1698.
[2] Gray, J., *Chin. med. J.* 1936, **50**, 761.

LATENT SQUINT AND THE AIRMAN

HETEROPHORIA may manifest itself in three forms: (1) as a simple clinical feature of ocular discord, or allied with a refractive error; (2) as a distress which appears while engaging in some occupation; (3) as a handicap in the achievement of a purpose. In the first group fall cases for the most part very definite in producing symptoms within the normal routine of life, requiring no binocular stress for their revelation. In general ophthalmic practice the difficulty is more often the result of hyperphoria, with or without some torsion, than of weakness of convergence or of esophoric excess. The second group may be considered as the by-product of industrial progress, whether it be the machinist or the keeper of ledgers that suffers. Here effort constantly repeated over long periods has undermined the ocular muscle system to produce, as a rule, some weakness of convergence. The third group, in which successful issue to a pursuit is frustrated, arises through an inability to estimate distance with the body moving fast, whether in a motor-car at ground level or in an aircraft above the earth. To say that the presence of certain degrees of latent squint does not exercise a very definite influence under circumstances of this kind is just as erroneous as to suggest that all cases in which the condition is found are incapable of dealing with these situations. In the assessment of heterophoria with respect to its influence upon judgment of approach in flying, fixed valuations act as guides, but are not infallible. They may be regarded as standards which apply to the

[3] Ezickson, W. J., and Feldman, J. B., *J. Amer. med. Ass.* Nov. 20th, 1937, p. 1706.
[4] Maitra, M. K., and Harris, L. J., *Lancet*, 1937, **2**, 1009, 1025.

great majority but to which, as in everything else clinical, there are exceptions. The complete explanation of any visual response is apt to lead into the limitless intricacies of psycho-physiology.

Take for example the convergence test as applied to flying personnel. There are various degrees in the response to this test.

1. Simple effortless convergence to around 6 cm.
2. Breakdown of convergence far out (12—16 cm.), but still without effort, in which one or other eye quietly loses interest and diverges.
3. Convergence forced by obvious effort to a standard distance (10 cm. or less).
4. Convergence forced, with general facial contortion and head-retraction, resulting in extreme ocular pain.
5. Complete loss of all desire to fixate from the moment the test object is moved towards the eyes.

The first case is the normal response. The second indicates a certain weakness of binocular accord in which under an exacting demand the judgment might be influenced by a predominance of one eye. This has been called by P. C. LIVINGSTON "dominant macular reception." In normal life no symptoms arise from it because, under conditions of near-vision concentration, the way of escape from binocular rivalry or confusion lies comfortably open. In aviation such cases have succeeded where the ability to judge distance is acquired through the same series of processes repeated over and over again in identical form. Difficulties are far more liable to arise where the pathway is disturbed by alternation; hence a monocular dominance gives a safer prognosis than one in which there is uncertainty as to the final pathway. The third response is the forced effort at convergence. Normally in such cases binocular life has been shared and SHERRINGTON'S final common pathway reached without discord. Some condition or set of conditions has however arrived making the act of convergence not a subconscious response but one in which some conscious force is demanded. Fatigue in its general sense is usually responsible. Such a state of affairs gives warning for a cautious ruling with regard to flying. Indeed flying strain itself may be the exciting cause of the condition, with an adverse effect upon consistent accuracy in landing aircraft. The fourth response is a later manifestation of the third. Here concentrative effort assumes authority over what was previously subconscious. There is behind these cases almost always a functional element, the structure of which rebels against a movement that has now assumed an active rôle. The normal pathway has been transferred elsewhere, the subconscious to the conscious, the natural to the artificial; and, as all things newly invoked are the more difficult of action, so much the greater is the energy demanded of a faltering will. In the fifth type of response the call to converge has lost its natural and harmonious rhythm and has become the servant of uncertain consciousness; the demand for energy in the act of convergence meets with no reply. Such a condition can arise only as part of a psychoneurosis, and the question as to flying ability is of course negatived.

A consideration of this one test—and there are others of equal importance—suggests an escape from the superficially apparent, as for example ocular muscle deficiency, to the far wider state embracing the nerve-supply to the muscles concerned, the interconnexions of those nerves within the brain, and the effector mechanism that drives the whole. If the symptomatic phorias are explored over this wide field fresh and much-needed light might be thrown on one of the potential dangers of flying.

LOCAL TREATMENT OF THE COMMON COLD

FOR better or for worse, as far as his confidence in his art is concerned, the modern doctor is aware that his clinical impressions of the efficacy of remedies are often fallacious. It is now recognised that there are few things in medical practice more difficult than to assess accurately and impartially the effect of any particular line of treatment. The pitfalls are especially numerous and deep in conditions such as the common cold, whose severity and duration vary not only in different people but in the same person in different attacks. Sometimes a coryza, beginning acutely with the initial symptoms dreaded by all of us, aborts and disappears within twenty-four hours. This is presumably because the virulence of the infecting organisms is low or, more probably, because the patient's resistance at the time is high; but the speedy cure is likely to be ascribed to any local treatment which has been used. The efficacy claimed for any "cure" is nearly always said to be contingent on its use at the earliest onset of symptoms, and few nostrums or applications profess to do much when once the attack is well established.

In recent years vasoconstrictors, containing adrenaline, ephedrine, or benzedrine, have been largely employed in the local treatment of coryza, their popularity having been greatly assisted by the well-planned advertisements of those interested in their sale. There is no reliable evidence that they can effect a cure; E. M. BOYD and W. F. CONNELL of Ontario, from experiments on students, conclude [1] that the topical application of benzedrine does not shorten the duration of a cold but that it relieves the symptoms resulting from nasal turgescence. Such relief is well worth while, provided that no harmful effects are caused. But it is not certain that such drugs may not, by inhibiting ciliary action and by admitting air more freely to the nasal passages, encourage the growth of the causative organisms. Some practitioners have an impression that infection of the sinuses is a more frequent complication of a cold when vasoconstricting agents have been employed. Again, antiseptics powerful enough to affect the organisms are likely to do damage to the delicate cells of the mucosa. On the other hand it must not be forgotten that the local application of these vasoconstricting drugs, or of mild antiseptics such as Argyrol, to the middle meatus is one of the recognised methods of treating incipient sinusitis.

The theory that the virus of coryza requires plenty of oxygen, and that Nature's attempt to

[1] *Amer. J. med. Sci.* December, 1937, p. 768.

cure is by blocking the nasal passages and asphyxiating the organisms, has led to the use of oily substances to coat the mucosa and exclude the air from them. E. P. POULTON [2] some years ago advised for prophylactic use a viscid oil (Glegg's mixture) which consists of three parts of paraffinum liquidum with one part of white vaseline with or without gr. ½ of menthol to the ounce, to be introduced not by a spray but with a pipette while the patient lies on his back. A. ORLEY,[3] having been impressed by the improvement which sometimes follows the instillation of lipiodol in cases of bronchiectasis and chronic bronchitis, has recently advised spraying the nares with oil in which a mild non-irritant antiseptic may advantageously be incorporated. J. R. ROBSON [4] agrees with POULTON that massive doses are essential and that spraying is useless and recommends Glegg's mixture with menthol.

The application of a bland oil has the advantage that it relieves discomfort and at any rate does no harm. Relief can also be obtained by syringing with a warm isotonic saline lotion at the risk of spreading infection, especially to the ears. If this method is used it must be with the least possible force; the patient must breathe through the open mouth and must cease syringing at once if there is any tendency to swallow or retch. As in otitis after bathing, it is by blowing the nose when it is full of fluid that the danger is greatest of forcing infected matter into the ears; the patient should be told not to blow the nose for a quarter of an hour after syringing, or at least to close only one nostril at a time while blowing gently down the other. Syringing gives most relief in the later "thick" stages of a cold, when it helps to remove the stiff mucous secretion. In young children the saline lotion may be dropped into the nostrils from a pipette. The common cold, with all its sequelæ in the way of infections of the nasal sinuses, ears, and lungs, is a formidable disease. For its prevention the most hopeful method is perhaps to attempt to diminish the sources of infection by dissuading the leisured public from spreading it unnecessarily and by educating employers to encourage workers to stay at home in the early infectious stage. The only method of aborting established colds, and even sinus infections, that seems to be gaining ground is physical rest and the administration of concentrated preparations of vitamins in full doses. Certainly no local treatment has as yet been found which contradicts the old statement that a cold runs its natural course in a fortnight but that, if carefully treated, it can be cured in two weeks.

ANNOTATIONS

A NEW PITUITARY SYNDROME

IT has long been a matter of surprise that so potent a hormone as vasopressin has not been implicated in disease. The failure to find a place for it in pathology does not come from want of trying. Many attempts have been made to isolate this hormone from blood and urine in hypertensive states; but, although success has been claimed from time to time, independent confirmation has never been forthcoming. It seems evident that if vasopressin is excreted in hypertension the ordinary methods of detection are too crude to detect it. Gilman and Goodman [5] have overcome this difficulty by concentrating urine and their finding that antidiuretic hormone could be demonstrated in the urine of dehydrated rats, provided the pituitary was intact, has given direct support to the view that the posterior lobe of the pituitary is active in normal metabolism. Hard on the heels of this work comes the recovery, reported elsewhere in this issue, of a pressor-antidiuretic principle, evidently vasopressin, from the urine of a patient who presented a strange combination of hypertension, hyperchromic anæmia, achlorhydria, and symptomless hyperglycæmia. This is an interesting development of the work of Dr. Noble and Prof. Dodds and their colleagues on the effect of vasopressin in animals. There seems little doubt that the association is not fortuitous, but an expression of overactivity of the posterior lobe of the pituitary, and it is likely that other cases of the same kind will be discovered in due course. The two papers we publish this week provide food for thought and indications for future research. They should stimulate fresh attempts to look for vasopressin in commoner forms of hypertension, for example, and they should cause the hæmatologist to inquire how a sadly overburdened gland like the pituitary can find time to concern itself with hæmopoiesis. Lastly, the medical philosopher will note here another instance of the tendency to break down the artificial barriers which the systematic text-books have erected—for the convenience of the student, no doubt, but to the detriment of understanding—between diseases. When a patient presents symptoms of more than one disease the association may be nothing more than accident. But to neglect the alternative possibility is an admission of mental amblyopia.

[2] *Lancet*, 1932, **1**, 933.
[3] *Brit. med. J.* 1937, **2**, 877.
[4] *Ibid*, p. 997.
[5] Gilman, A., and Goodman, L., *J. Physiol.* 1937, **90**, 113.

THE DANGERS OF THOROTRAST

IN some quarters there is a growing disposition to restrict the use of Thorotrast to urgent cases in which no other diagnostic measure has provided a substantial clue, but elsewhere this substance is still being injected into patients far too recklessly. The fact that it is radioactive and is retained in the reticulo-endothelial system for an indefinite time should give pause to the more enthusiastic of its users. Taft,[1] using the Geiger counter which is sensitive to minute quantities of radiation, has shown that one clinical dose of thorotrast (75 c.cm.) gives the gamma-ray equivalent of 1·37 microgrammes of radium. When it is recalled that 2 microgrammes of radium have produced symptoms of radium-poisoning it will be seen that the danger from injecting such large volumes of thorotrast is serious. The doses used, if it must be used, should be the smallest that will give the desired radiographic effect.

Apart from risks contingent on the radioactive properties of thorotrast it has now been shown by

[1] Taft, R. B., *J. Amer. med. Ass.* 1937, **108**, 1779.

Lanari, Jorg, and Aguirre [2] that this substance causes a slowly progressive fibrosis of the liver accompanied by fibrosis and the formation of Gandy-Gamna nodules in the spleen; from which they conclude that it should not be used as a diagnostic agent. In neurosurgery thorotrast is most often employed to outline the vessels of the brain by intracarotid injection. Doubtless, if the amounts injected are small, this procedure is seldom detrimental to the patient. It has however been shown by Northfield and Russell [3] that the thorotrast may sometimes occlude small veins and capillaries in the neighbourhood of compressing tumours or abscesses; such occlusion may not only aggravate symptoms but hinder recovery when the tumour has been removed. Thorotrast has been used, too, in ventriculography, the injection being made into the ventricular system, and the histological effects of this procedure have lately been investigated by Freeman [4] in material from two biopsies and eight necropsies. The longest interval after injection was two months. He finds microscopic evidence of inflammation of the choroid plexuses and of the ependyma. In the meninges leucocytes and macrophages emerge early in response to the presence of this foreign substance. Later the leucocytes tend to disappear while the macrophages become more numerous. Yet the thorotrast appears to remain in the meshes of the pia-arachnoid without exciting much chronic inflammatory reaction. At the end of two months Freeman notes a perceptible thickening of the meninges about the base of the brain with a large proportion of the thorotrast still lying free in the spaces. His suggestion that this causes no blockage of the cerebro-spinal pathway will hardly pass without challenge, and it is startling to find that his observations do not deter him from continuing to use thorotrast in this way. Others will feel that injection of thorotrast into the ventricles must now be considered so dangerous as to be unjustifiable.

PRECORDIAL LEADS IN ELECTRO-CARDIOGRAPHY

IT is already clear that in myocardial infarction electrocardiograms taken with chest leads are of value in supplementing the information given by the limb leads. There is also some evidence to suggest that they will be helpful in other cardiac conditions, and it seems probable that in the future no electrocardiogram will be considered complete unless it includes at least one curve taken with a chest lead. It is even conceivable that the latter will supersede the curves obtained from the limbs. At present, however, there is divergence of opinion on the polarity and the site of the electrode applied to the chest. The most convenient method appears to be the one in which the deflections are in the same sense as those of the limb leads, and the normal T wave is upright. The left-arm electrode is placed over the heart, and that on the right arm is the indifferent electrode—the fact that this will now produce a negative instead of a positive deflection as compared with limb leads I and II does not seem an important objection. The exact site of application of the electrode to the chest wall must be specified, since a slight alteration in its position can cause considerable change in the form of the curve. De Ruyter and Bechhoff [5] go so far as to apply their electrodes over certain fixed points of the cardiac profile while the patient is under fluoroscopic observation. They take four points anteriorly, which may be roughly described as corresponding to the four corners of the heart, and four on the posterior chest wall at similar levels. To these eight points a pair of electrodes may be applied in 56 different ways, giving 56 different curves, but 28 of these are inversions of the remainder. A special frame is used carrying movable electrodes and this is lowered over the patient and the eight electrodes are applied at once. It was found that usually with antero-posterior leads the curve was much the same whichever posterior point was used, so that in normals four dorso-ventral leads were sufficient. In the presence of cardiac enlargement, however, curves taken from a variety of dorsal points may have special value. Curves are shown obtained from 12 different chest leads, both normal and from cases with each variety of ventricular predominance. It is clear that such a multiplicity of chest leads is impracticable as a routine procedure. Further work will be necessary before the best site for applying the precordial electrode can be decided and also whether the site used must be defined so precisely as to require fluoroscopy.

THE MENTAL EFFECTS OF ALTITUDE

ALTHOUGH many writers have drawn attention to the mental effects of great altitudes, no careful psychological studies have been published of the mental states of climbers in the high Himalayas. It would clearly be impossible adequately to disguise the identity of the patients whose symptoms were described. We know enough, however, to realise that mental changes due to oxygen-lack can seriously affect the chances of success on Everest. Irritability is common. Occasions have been described on which a tired man has confused the identities of other members of the party. A state bordering on persecution mania may develop. On one occasion a climber was carried for several days down a great mountain because his "frost-bitten" feet made walking "impossible." His feet on examination were found to be perfectly normal, but his belief in his pain was genuine. The older experiments in low-pressure chambers were often interrupted by the curious behaviour of the subjects, but their symptoms were seldom studied in detail.

Gellhorn and Kraines [1] of Chicago report experiments on the associations of normal medical students subjected to oxygen-want, increased carbon-dioxide percentage, and hyperpnœa. The mental associations were studied by the method of Kent and Rosanoff: this measures not the speed of association, which in these conditions has already been shown by Lowson and Wespi to be reduced, but the "unusualness" of association. In all the three conditions examined there was an increased "unusualness" of response to given stimulus words, or, as the authors put it, a "shift to the left" in the Kent-Rosanoff test. There was also an increase over the control group in the phenomena of "perseveration," or the repetition of the same word to several stimuli; and of "dissociation," or reaction for which neither experimenter nor subject could find any explanation. The three classes of abnormal response become more frequent as the concentration of oxygen falls. Concurrently there was an increase in dizziness and tremor and a

[2] Lanari, E. C., Jorg, M. E., and Aguirre, J. A., *Pr. méd.* Nov. 6th, 1937, p. 1562.
[3] Northfield, D. W. C., and Russell, D. S., *Lancet*, 1937, **1**, 377.
[4] Freeman, W., *Arch. Neur. Psychiat. Chicago*, 1937, **38**, 346.
[5] De Ruyter, F., and Bechhoff, J. E., *Scalpel*, Oct. 23rd, 1937, p. 1476.

[1] Gellhorn, E., and Kraines, S. H., *Arch. Neurol. Psychiat., Chicago*, 1937, **38**, 491.

decrease in acuity of vision and hearing, as described by earlier investigators.

The interest in this work lies chiefly in the possibility of a correlation with similar tests performed in cases of mental disease. The tendency to unusual or individual response and to perseveration is naturally greater amongst the insane, and, when very prominent, is believed by Kent and Rosanoff to be pathognomonic of schizophrenia. But until the tests have been conducted under more stringently controlled conditions and in a far larger number of mental states it is impossible to gather any sound conclusions from them.

EMBOLISM UNDER OBSERVATION

HITHERTO most of our knowledge of the effects of embolism has been gathered from the study of dead tissue, and we have little information about the reactions that take place in blood-vessels during life when they are occluded by solid material. Some important observations are now described by Villaret, Cachera, and Fauvert [1] who have succeeded in demonstrating changes in the vessels of the pia mater after embolism both solid and gaseous. Using Forbes and Wolff's technique they were able to view these vessels in the living state in dogs through trephine openings in the cranium, and to obtain serial photographs of the changes following injections into the carotid artery of watery suspensions of pumice-stone containing angular particles about 150 μ in diameter. They record some remarkable reactions in the arterioles not only at points where particles became impacted, but also elsewhere. First there was a local vasoconstriction at the site of impaction, producing a fairly uniform area of ischæmia in the tissues supplied by the affected vessel, but later a series of peculiar narrow spasmodic constrictions took place in the arterioles both above and below the site, and often at considerable distance from it. The investigators show a number of photographs illustrating these constrictions. They appear as sharp indentations in the arteriolar walls, sometimes sufficient almost to occlude the lumen; they occur characteristically about bifurcations, often affecting the larger of the two branches, as if they were designed to divert the stream into the other branch; and they develop fairly rapidly, attaining their maximum in a minute or two, and often disappearing to recur again, the whole phenomenon sometimes lasting for half an hour or more.

The findings with gas emboli, on the other hand, were entirely different. Even bubbles of air large enough to fill the lumen of the arteriole and to arrest the circulation seemed to give rise to no reaction whatsoever. There was no constriction of the vessel, but rather a tendency to flaccid dilatation. The arrest of the circulation was as a rule only temporary, the air bubbles being gradually forced through to be dispersed in the capillary circulation and never to reappear again in the veins. Raising of the blood pressure by adrenaline accelerated this dispersion, but yielded no evidence of vasoconstriction such as was seen with solid emboli. Villaret and his colleagues are content to record these findings without venturing on deductions, but they hold that the phenomenon of arteriolar spasm, which has long been postulated in neurology, has now been verified and actually demonstrated, as a reaction produced by direct local stimulation. What part this plays in the development of tissue degeneration cannot yet be decided, but their findings with regard to gas embolism incidentally serve to disprove the hypothesis that vascular spasm explains the nervous disorders often been ascribed to this accident. Apparently blood-vessels are indifferent to the presence of gas in their lumen.

[1] Villaret, M., Cachera, R., and Fauvert, R., *Pr. méd.* 1937, **45**, 1555.

PERITONEOSCOPY

WHEREAS the use of an optical instrument to allow pleural adhesions to be divided under direct vision is a recognised procedure, peritoneoscopy has so far met with no sort of general acceptance. The difference is, of course, that an exploratory operation is far more formidable in the thorax than in the abdomen. Ruddock [1] has used the method in 500 cases referred to him from a large general hospital and from private physicians. He considers that though peritoneoscopy should not replace the diagnostic laparotomy, in many cases it is the procedure of choice. The main point he produces in its favour is that it requires such a small incision that patients need not be kept in bed or have a restricted diet. In acute disease peritoneoscopy is contra-indicated, but in many chronic conditions Ruddock has been able to obtain all the information necessary through the very restricted opening. It is possible, for example, to decide definitely the presence of secondary deposits of carcinoma in the liver when operation is contemplated for carcinoma of the stomach. Where the cause of free fluid in the peritoneal cavity is in doubt, the appearance of the peritoneal surface may settle the diagnosis. The method also enables biopsy specimens of tumour masses and tissues to be safely and painlessly removed. The patient is thus saved the risk of laparotomy, which Ruddock puts as high as 6 per cent.

The examination is conducted under local anæsthesia with Novocain, ¼ grain of morphia being the only premedication. A pneumoperitoneum needle is first inserted and the cavity blown up with air; where the peritoneum is already under tension from ascitic fluid this preliminary inflation is not necessary. A sheath with a bistoury-pointed obturator is next inserted, the puncture being carried out steadily and cautiously with the instrument pointing to either side of the spinal column. The actual site of puncture is a matter of convenience and is determined by the area to be examined. After effecting an entrance the obturator is removed and a telescope inserted, very much like that of a cystoscope. Any fluid present is evacuated and replaced by air—the only sensation this produces is one of fullness. By elevating different parts of the abdomen and pelvis the air is shifted and the intestines moved out of the way, allowing space for manipulating the peritoneoscope and light for examining the surfaces of the organs. Inflation of the stomach with air makes it appear luminous. The Trendelenburg position brings the pelvic organs into view. The liver surface seems to have been very successfully examined. The biopsy forceps, after removal of a piece of liver or other solid tissue, are made to act as an electrode and the raw area sealed by cautery. The only death recorded in the 500 cases was due to fatal hæmorrhage from a metastatic nodule in the liver. At the end of the examination the air is allowed to escape and the instrument is removed. A skin clip or stitch closes the perforation. There were no herniæ through the site of puncture. Perforation of the intestine occurred in 8 patients, in all of whom the bowel was firmly

[1] Ruddock, J. C., *Surg. Gynec. Obstet.* November, 1937, p. 623.

plastered against the parietal wall; Ruddock believes that it is only in these cases that it can be punctured. No serious consequences follow, since the general peritoneal cavity is not infected.

THE ANÆMIA OF IRON DEFICIENCY

C. W. Heath and A. J. Patek [1] have recently brought forward interesting additional evidence on the ætiology of so-called idiopathic hypochromic anæmia, based upon the personal study of over 300 cases of various types of anæmia responding to iron. Their conclusion is that idiopathic hypochromic anæmia is due to iron deficiency and can be relieved by adequate iron therapy. The cause of the iron deficiency is almost invariably chronic blood loss, commonly from bleeding hæmorrhoids, slightly excessive menstrual loss or epistaxis. In a series of 60 cases of apparently typical idiopathic hypochromic anæmia only 7 failed to show some pathological loss of blood, and in these for various reasons it was impossible to make complete and controlled studies. Further, patients whose blood loss could be checked did not need maintenance treatment with iron; while in those who continued to bleed the administration of iron had to be continued indefinitely to maintain a satisfactory blood picture. Diets poor in iron, intestinal malabsorption associated with achlorhydria and multiple frequent pregnancies play a secondary but important rôle in preventing repair of the blood. The ætiology of the achlorhydria which Heath and Patek found in 45 of their series of 60 cases remains unexplained and is idiopathic in the sense that it often appears to be constitutional or inherited in many cases. Diets poor in iron were present in only 37 per cent. of the 60 cases studied. Heath and Patek, in confirming the impression of many workers that blood loss in some form is the essential factor in the ætiology of idiopathic hypochromic anæmia, have apparently destroyed any justification for the term "idiopathic." They do not, however, suggest that it should be discarded or that such cases should be grouped with the more obvious post hæmorrhagic anæmias. The clinical picture they represent is a distinct entity, typically a long-standing anæmia, usually in middle-aged women, associated with achlorhydria or hypochlorhydria, atrophic glossitis and koilonychia; the blood loss is usually not at first apparent. To what extent a universal diet really adequate in iron content would prevent the occurrence of this type of anæmia in women must be a matter of speculation. But it seems clear that for the moment anyhow "idiopathic" hypochromic anæmia can be regarded as a conditioned deficiency—the conditioning factor being blood loss.

EUPHYLLIN IN CARDIAC DISEASE

Theophylline ethylenediamine (more familiar under the names of Euphyllin, Aminophylline, and Metaphyllin) has been reported to dilate the coronary vessels experimentally,[2] and has been used in the treatment of angina. It is also being administered as a diuretic and cardiac tonic in heart disease with every variety of failure, and in combating Cheyne-Stokes respiration. For the relief of any kind of cardiac pain the value of this drug (like that of the other purine derivatives) is questionable, even if it is admitted to have a powerful coronary dilator effect in animals, and there is not complete agreement on this.[3] Conclusions about the merits of such a remedy must be drawn from clinical studies, and a number of workers [4 5] have found euphyllin of no use in the routine treatment of angina. Brown and Riseman [6] record an increase in exercise tolerance after administration of euphyllin and other theophylline preparations in 17 patients with angina, but gastric symptoms (nausea and heartburn) often arose while the drugs were being taken, and when severe they discounted any favourable action on the cardiac pain. An increase in exercise tolerance of 50 per cent. or more was demonstrated in only 4 or 5 patients. de Balsac [7] reports the results of giving euphyllin in 30 cases with cardiac symptoms of various kinds, but as he admits, the results are inconclusive. With daily doses exceeding half a gramme a liability to headache, insomnia or excitement, and gastric discomfort was noted. It is clear that euphyllin has certain effects on the brain, and its ability to abolish Cheyne-Stokes respiration has been known for some years. According to Marais and McMichael [8] this action comes from stimulation of the medullary respiratory centres, and is chiefly due to the ethylenediamine component. In patients with early cardiac failure Starr and co-workers [9] found euphyllin capable of increasing cardiac output as estimated by the ethyl iodide method, and on its value as a diuretic in cases of failure with œdema there is fairly general agreement, some holding that it is more effective than any other purine derivative. It may be concluded that it is a powerful drug which may relieve symptoms of congestive heart failure, but that its efficacy against any type of cardiac pain remains unproved.

AN INERT EXCRETION PRODUCT OF PROGESTERONE

PROGESTERONE is an irritating hormone. It has been isolated from luteal tissue, its structure has been determined, it has been synthesised from stigmasterol, and it has been shown to play an essential part in the menstrual cycle and in pregnancy. But it has hitherto obstinately resisted repeated attempts to recover it from urine and blood. In this respect progesterone contrasts sharply with the gonadotropic and œstrogenic hormones, which during pregnancy are discarded so lavishly in the urine that this despised fluid is now a valuable commodity, coveted as much by the western manufacturing chemist as it always has been by the oriental peasant. Thus it is possible, even with the present rough methods of biological assay, to detect significant changes in the output of prolan and œstrin in certain diseases. But without parallel information about the secretion of progesterone, which is at once complementary and antagonistic to œstrone, it is impossible to interpret these changes. To take an instance, there is evidence that the output of œstrin tends to be low in eclamptic toxæmia as compared with normal pregnancy. This may be a primary defect. It may equally well be a compensatory adjustment to a primary alteration in progesterone secretion. In the one case œstrin is indicated as a therapeutic measure; in the other it is contra-indicated. The clinician, in fact, has no means of judging whether

[1] Heath, C. W., and Patek, A. J., *Medicine*, 1937, **16**, 267.
[2] Fowler, W. M., Hurevitz, H. M., and Smith, F. M., *Arch. intern. Med.* 1935, **56**, 1242.
[3] Wiggers, C. J., and Green, H. D., *Amer. Heart J.* 1936, **11**, 527.
[4] Evans, W., and Hoyle, C., *Quart. J. Med.* 1933, **2**, 311.
[5] Gold, H., Kwit, N. T., and Otto, H., *J. Amer. med. Ass.* 1937, **108**, 2173.
[6] Brown, M. G., and Riseman, J. E. F., *Ibid*, 1937, **109**, 256.
[7] de Balsac, R. H., *Paris méd.* Nov. 27th, 1937, p. 423.
[8] Marais, O. A. S., and McMichael, J., *Lancet*, 1937, **2**, 437.
[9] Starr, I., et al., *J. clin. Invest.* September, 1937, p. 799.

œstrin therapy is likely to be helpful, harmful, or merely futile, unless he is prepared to submit his patient to the risk of empirical trial. For such reasons much interest attaches to the recent work of E. H. Venning and J. L. S. Browne [1] on a substance, pregnanediol, which appears to be an excretion product of progesterone. Pregnanediol is biologically inert, but resembles progesterone in structure. It appears in the urine in considerable amounts during pregnancy and to a less extent during the luteal phase of the menstrual cycle. It is also excreted after intramuscular injection of progesterone. It can be recovered quantitatively by extraction with butyl alcohol and measured gravimetrically as sodium pregnanediol glucuronidate. It seems fairly certain, then, that progesterone is normally converted into this substance for excretion, and estimation of the output of pregnanediol should therefore throw light on the secretion of the luteal hormone. Application of this work to the study of disease is not likely to be delayed for lack of material. Progesterone deficiency has been suspected as a cause of one type of habitual abortion. It has also been suggested by J. M. Rolson and S. J. Paterson [2] and, independently, by J. Young [3] as a possible factor in eclampsia. Irregularities of menstruation provide a further group of complaints in which information about the activity of the corpus luteum is clearly desirable. The work of Venning and Browne promises to throw light on these and other gynæcological problems of practical importance.

PENTOTHAL SODIUM

THE production of barbituric compounds appears to know no bounds and as each new drug arrives it is heralded as an improvement on the last. This claim, naturally enough, is not always borne out by further experience, and already some of the earlier compounds are dropping out of use. At present the running, so to speak, is being made by Pentothal sodium which is declared to be an improvement even on Evipan. The newer body, it is said, produces no excitement—an unusual merit in a barbiturate—induces unconsciousness more quickly, and less often causes jactitation. Good accounts are given of its use in childbirth, and in Toronto Wesley Bourne and others [4] have given it to 238 women. Observations made by Bourne show that the duration of labour is shortened by its use, but that its amnesic effect is slight. Especial care must be taken during the intravenous injection not to let any of the liquid escape into the surrounding tissues, for there its power for harm seems to be considerable. Brown and Troup [5] in Australia are also well pleased with pentothal and record an anæsthesia of 25 minutes without adjuvant. The drug is generally used either for short operations or as an introduction to the anæsthetics with a longer action. It is employed as a 5 per cent. solution. The Toronto workers when using it for labour confine it to the early stages, going over to nitrous oxide and cyclopropane later. They are also enthusiastic about the value of Vinyl ether for combating the pains of the later stages of labour, after pentothal has been used earlier, and think that this agent (vinyl ether) particularly helpful in general practice. Pentothal sodium is being widely tried in this country too, and we recently published the experience of Mallinson [6] who gave a general account of the technique required, including Jarman's method [7] for continuous use of the drug in long operations. It is still questionable whether any barbiturate employed in this way is to be preferred to the other drugs more usually applied in major surgery.

[1] *Endocrinology*, 1937, **21**, 711.
[2] *Brit. med. J.* 1937, **1**, 311.
[3] *Ibid*, p. 953.
[4] MacPhail, F. L., Gray, H. R. D., and Bourne, W., *Canad. med. Ass. J.* November, 1937, p. 471.
[5] Brown, G., and Troup, G., see *Med. J. Aust.* Oct. 16th, 1937, p. 687.
[6] Mallinson, F. B., *Lancet*, 1937, **2**, 1070.
[7] Jarman, R., and Abel, A. L., *Ibid*, 1936, **1**, 422 and 600.
[8] See *Lancet*, 1936, **2**, 328.

WHO PAYS THE DOCTOR CALLED TO AN ACCIDENT ?

A Good Samaritan wrote to the *Times* last week in protest against his liability to pay for the professional services he procures in an emergency. He had seen a man borne into a chemist's shop bleeding and unconscious after a fall in the street. He had himself summoned the nearest doctor. Afterwards he learnt that, by the fact of having discharged this humane duty, he might have been deemed to have employed the doctor and thus might have made himself responsible for a fee. A police inspector, with whom the Good Samaritan discussed this surprising position, confirmed the obligation: the person who calls in the doctor, like the person who calls up the fire brigade, may be expected to pay the bill. Perhaps, as the Good Samaritan says, this is quite wrong. But before sympathising too warmly with his plaint we would wish to know how often in practice the caller is dunned for a medical fee; also whether in an emergency the impulse to find a doctor, whether it arises from self-importance or from genuine concern, would really be checked by the fear of having to pay him. We hear, too often, the other side of the story; of the country doctor, just settling to a meal after surgery, summoned to the cross-roads 3½ miles away to an accident of which no trace can be found by the time he reaches the spot; of a protest at the demand for a fee of £1 1*s.* for a night visit involving first aid and conveyance of a bleeding patient to hospital. It is without any regard for the doctor's point of view that the Good Samaritan continues: "Surely it should be regarded as a legal (as it is already a moral) duty for the nearest doctor to attend in the first instance upon any emergency call and afterwards to hand over the patient to his own medical attendant."

The idea underlying this letter may be that the services of a surgeon should always, like those of the police or the scouts of the A.A. or R.A.C., be available in emergency. The mechanism of such a scheme is left to the imagination. The provision of a rota of hospital doctors awaiting calls to outside accidents is a method that seems to work well in Russia,[8] and will perhaps prove to be the solution of the problem in other countries. It would certainly ensure that the patient, having been "rushed" to hospital, would not have to wait for skilled attention when he arrives there. At present the nearest practitioner is expected to neglect his duties to his regular patients at a moment's notice, and to let no thought of any sordid element of money or of fatigue prevent him from obeying a summons which may be quite unjustified or even frivolous. The Good Samaritan in the parable deposited his twopence and undertook to repay whatsoever more might be expended on the patient. His modern counterpart makes no mention of any payment by anybody. A suggestion has reached us that the cost of any scheme could be met by levying entertainment tax on the crowds which habitually gather round any street accident. Seats

(suitably roofed over for promising wet days) might be let at good prices near any dangerous corner, where already the neighbouring residents are prone to assemble hopefully.

HEALTH OF THE SCHOOL-CHILD

IT is a sign of the times that nutrition occupies the most prominent place in the annual report for 1936 of the chief medical officer of the Board of Education.[1] Though it has long been realised that a child's body must be adequately nourished before it can derive full advantage from mental and physical education, the supplementary nourishment provided at some schools has not in the past been adapted to the needs of the individual. The Board of Education, taking account of modern discoveries in physiology, have now emphasised, in Circular 1443, their anxiety that the free meals supplied should not be "of the soup kitchen type" but appropriate to the deficiency; also that it is the duty of local authorities not to rely on applications from parents for the discovery of undernourished children but to invite reports from members of the school medical service or the public health service, from teachers, school nurses, and attendance officers, and from others in regular contact with the children from day to day. It is specifically stated that "any educational evidence, any inability to profit from mental and physical instruction, any lassitude, however slight these may be, may justify provision" of extra food.

The rest of the report describes progress in physical training with special consideration of the duties of organisers; medical inspections at schools and at junior instruction centres; various forms of treatment arranged through the agency of the school medical service; nursery schools and administrative measures for the welfare of the young child; the development of the school dental service, with special reference to the influence of diet on caries in children's teeth; the teaching of hygiene in schools; the education and training of the blind; and the provision of holiday camps. The work done by local education authorities and voluntary agencies in this last respect has led to such good results that the provision of camps on a much more extensive scale would be welcomed.

BISMUTH POISONING

TWENTY-FIVE years ago, when bismuth subnitrate was used in gastro-intestinal radiography and as an application to wounds, poisoning was not infrequent; but nowadays, despite the wide employment of bismuth in antisyphilitic treatment, serious toxic symptoms are rare. The more soluble salts of bismuth resemble those of mercury and the other heavy metals in causing stomatitis, gastro-enteritis, and nephritis, the stomatitis due to bismuth showing characteristic blue-black areas on the gums. Bismuth salts tend particularly to affect the kidneys and to produce a rapidly developing oliguria, but permanent renal damage is uncommon. Dowds [2] has reported three cases in which death followed 2–4 weeks after intramuscular injection of about 25 grains of sodium bismuth tartrate, the symptoms being oliguria and stomatitis; post-mortem examination showed congestion of the renal cortices and fatty liver. Even where there is almost complete anuria, however, recovery is the rule. Thus Keith and Osterberg [3] describe two cases in which a solution of bismuth and potassium tartrate was introduced into the urinary tract for radiographic purposes. One patient had a chronic pyelonephritis and an enlarged prostate and the other a ureteral stone. Both developed oliguria and vomiting within 24 hours of the introduction of the solution and an ulcerative stomatitis 3–4 days later. The blood-urea of both rose rapidly and remained high for about three weeks, but they then recovered and they were in excellent health three years later. In these cases it is possible that the effects were partly due to more highly toxic bismuth compounds present in the solution. That especial care with dosage must be taken with sickly patients is demonstrated by Eitzen's [4] case of a frail boy of 8 years who was given an injection of "thiobismol" by an osteopath as a highly original treatment for sore-throat. He passed no urine for the next six days and very little for the following week, but recovered completely within two months. Here, apart from being unnecessary, the dose given was too large. Of other symptoms, methæmoglobinuria has often been recorded in poisoning by bismuth subnitrate, and Lueth [5] has described a case in which prolonged use of this drug caused blue-grey pigmentation of the skin. Death from "colloidoclastic shock" has occasionally followed an injection within a few minutes, possibly from the drug being passed into a vein by mistake.

It is doubtful whether sodium thiosulphate has any effect as an antidote, and where there is interference with renal function the most satisfactory measure has been intravenous saline and glucose. The oily suspensions of bismuth appear to be the safest preparations, and the intravenous route is definitely to be avoided.

MEDICAL OXFORD AND THE DOMINIONS

Lord Nuffield has offered to the University of Oxford a further sum of £168,000 to provide more facilities for graduate students from South Africa, Australia, and New Zealand to share the advantages of the Oxford school. It is proposed to establish three demonstratorships to be held in turn in the departments of anatomy, biochemistry, pathology, pharmacology, and physiology, and three assistantships to be held in the departments of medicine, surgery, obstetrics and gynæcology, anæsthetics, orthopædic surgery, and therapeutics. Universities of Australia, New Zealand, and South Africa which grant medical degrees will in turn select holders from among their graduates who already hold research posts or who are eligible for them. It is also proposed that the university should appoint from time to time a visiting professor who would tour the Dominions to give information about the facilities for research in Great Britain and to find out the needs of the Dominions.

Lieut.-Colonel A. StG. Mallam, Royal Army Medical Corps, has been awarded the O.B.E., and Captain G. E. S. Stewart, Indian Medical Service, the M.B.E., for their services during the operations in Waziristan from Jan. 17th to Sept. 15th, 1937.

AT its meeting on Dec. 15th the council of Leeds University instituted a lectureship in medical hydrology and appointed Dr. Geoffrey Holmes of Harrogate to the post. This is, as far as we know, the first time that medical hydrology has been officially placed on the curriculum of study of any medical school in this country.

[1] Annual Report of the Chief Medical Officer of the Board of Education for 1936. H.M. Stationery Office. Pp. 136. 2s. 2d.
[2] Dowds, J. H., *Lancet*, 1936, **2**, 1039.
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SPECIAL ARTICLES

SAFETY FACTOR OF DESIGNATED AND OTHER TYPES OF LIQUID MILK

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The medical practitioner is so often consulted by his patients about the milk-supply that it is important for him to have clear ideas as to the different kinds of designated milk—both what they aim to be and their safety as human food, particularly for the child. This short article endeavours to describe the types and their safety factors, as clearly as possible, as an aid to the busy practitioner. To keep it as short as is practicable I have set out the information somewhat dogmatically; but the evidence for every statement is readily available in numerous publications and much of it will be familiar.

DISEASES SPREAD THROUGH MILK

(*a*) *Acute notifiable infectious diseases.*—Scarlet fever, diphtheria, paratyphoid fever, and enteric fever have frequently been spread by milk; bacillary dysentery and acute food poisoning rather rarely. Measles, whooping-cough, small-pox, and chicken-pox have never been spread by milk.

(*b*) *Streptococcal sore-throat.*—Outbreaks of streptococcal sore-throat spread by milk are comparatively rare but may be widespread, as in the Brighton-Hove outbreak with over 1000 cases and more than 60 deaths. In 1905 I had to try to control such an outbreak with over 600 cases.

(*c*) *Tuberculosis of bovine origin.*—With the considerable extension of pasteurisation the number of cases is steadily diminishing but even now we can hardly put the deaths at less than 1700 per annum, with about 4000 infections.

(*d*) *Undulant fever.*—The number of fresh cases each year is considerable but there are no accurate returns. Prof. G. S. Wilson estimates that there are about 500 cases annually but some are direct infections from diseased cows and not actually milk-spread.

(*e*) *Indefinite infections such as gastro-enteritis.*—There is definite evidence that such cases occur but our information is inadequate. With the recent recognition of the fact that certain staphylococci and streptococci produce toxins which cause definite gastro-enteritis this factor cannot be ignored, but probably it is not of great importance.

For more detailed information on milk-spread disease see Savage (1937).

THE PRESENT OFFICIALLY RECOGNISED TYPES OF MILK

In the following list those marked with an asterisk are the graded milks prescribed by the Milk (Special Designations) Order, 1936.

(1) *Tuberculin-tested milk* * is produced from herds whose cows are free from tuberculosis and are maintained free from tuberculosis by a system of veterinary inspections with tuberculin-testing of each animal not less than once every six months, and a system of isolation. There is also a bacteriological standard of cleanliness and requirements as to hygienic production. If milk so produced is bottled on the farm it can be described as *Tuberculin-tested Milk (Certified)** ; otherwise the conditions are the same. It may also be pasteurised under the approved official conditions; it then has to be called *Tuberculin-tested Milk (Pasteurised)** and the bacterial standard is much higher (30,000 bacteria per millilitre).

(2) *Accredited milk.** —This is milk from ordinary cows (i.e., there is no tuberculin-testing) but the cows are subjected to a quarterly clinical veterinary examination. On the other hand, there is no restriction as to the admission of any cows at any time apart from the admission of a known tuberculin reactor. The main requirements are of much more hygienic production (the standards enforced varying very greatly in different areas) and a bacteriological standard of cleanliness which is the same as for tuberculin-tested milk.

(3) *Pasteurised milk* * is milk which has been retained at a temperature of not less than 145° and not more than 150° F. for at least thirty minutes and then immediately cooled to not less than 55° F. The plants used and the conditions of working are controlled by the local authorities who grant the licence, while certain requirements are prescribed such as the use of automatic temperature records. There is a bacteriological standard for any time before delivery to the consumer, but no standard as to the quality of the milk to be pasteurised.

(4) *Ordinary milk* is subject to no bacteriological standards or any standards as to freedom from pathogenic organisms. The only control over its production is the supervision exercised by the local authorities under the Milk and Dairies Order, 1926. The requirements of the Order itself are vague in many particulars and the enforcement (except in urban areas where there are very few cowsheds) is very unequal and in many areas nominal.

RISK OF TRANSMISSION OF DISEASE

The risk of contracting disease from milks of the different types is conveniently demonstrated in the following Table which shows how far this risk has been removed by the precautions taken.

Human disease.	Raw milk.	Tuberculin-tested.	Tuberculin-tested (pasteurised).	Accredited.	Pasteurised.
Acute infectious diseases..	+ + +	+ +	−	+ +	−
Sore-throat outbreaks ..	+ + +	+ + +	−	+ + +	−
Tuberculosis	+ + +	−	−	+ +	−
Undulant fever	+ + +	+ + +	−	+ + +	−

+ + + = Full risk. + + = Risk somewhat reduced.
+ = Risk greatly reduced. − = Risk removed.

In preparing this Table I have taken the view that the better general control over production which is required for tuberculin-tested and accredited milk is of some slight value in reducing the risk of infectious disease transmission; but inasmuch as a survey of the health of the milkers is no part of the conditions of production many will not accept this view and will hold that there is no protection at all. I have given these milks the benefit of the doubt.

As regards sore-throat outbreaks, which are caused by highly specific streptococcal infections of the cow's udder, not capable of detection by veterinary inspection and having nothing to do with ordinary cow mastitis, I do not think there is any evidence

that the veterinary inspection required is likely to lead to a reduction of incidence.

The extent to which the quarterly clinical veterinary inspection required for accredited milk reduces the likelihood of tubercle bacilli being present is a matter of considerable interest. It has to be remembered that the ordinary accredited herd can and often does contain just as high a proportion of cows affected by tuberculosis (as shown by a positive reaction with tuberculin) as the ordinary herds. Whether or not tubercle bacilli are being excreted depends upon the site of the infection, and at any time such a cow may become an "open" case actively eliminating tubercle bacilli. Every veterinary surgeon of experience agrees that these animals can be detected only by the use of the tuberculin test, which is *not* used for accredited milk. In fact it is not at all uncommon for the bacteriologist to find tubercle bacilli in the milk of a herd and for the most experienced veterinary surgeon to be unable to detect the animal by clinical examination; he may only be able to do so by a long procedure of bacteriological testing of the milk of individual cows and groups of cows. Furthermore herds are continually changing and additions taking place between the quarterly examinations, and these additions may include infective animals. It is not possible therefore to rate very highly the value of this examination as regards protecting the milk from infection with the tubercle bacillus. It is possible however to give a quantitative estimation of the risk reduction. Statistics obtained by the People's League of Health showed that out of 5274 samples of Grade A milk (the old name for accredited milk) 5·59 per cent. contained tubercle bacilli. For ordinary raw market milk (not bulked milk, the percentage infection of which is 100) the proportion containing tubercle bacilli is 6–7 per cent. Taking a figure of 6·5 per cent. a reduction to 5·59 represents a percentage reduction of 14. I think it is a fair calculation to say that the quarterly clinical veterinary examination affects a reduction of risk of from 14 to at the most 20 per cent.

The Table is based upon the assumption that the pasteurisation is properly controlled and performed. Unfortunately the Milk Designations Order gives the control to the rural and urban councils and this duty is not adequately carried out everywhere. Where, as for example in Somerset, a proper system is followed and adequate laboratory controls are used pasteurisation is very efficient. The phosphatase test gives complete laboratory control when combined with suitable inspection.

OBSERVATIONS

The Table shows very clearly that only pasteurised milk is really safe from the risk of conveying disease. Given efficient pasteurisation, with the milk bottled and thus secure from the possibilities of subsequent infection, the safety factor is absolute.

The Table is a little misleading in one way, because it cannot bring out the *relative* importance of the risks run. Tuberculosis is by far the most important condition so that a definitely higher value attaches to tuberculin-tested milk than is apparent in the Table. The elimination of the risk of conveying tuberculosis through milk is such a great advance that it is reasonable to attach great importance to this type of milk even if it is not pasteurised. When I was county M.O.H. for Somerset the only milks allowed to be used for school-children without boiling were pasteurised and tuberculin-tested.

In July last the Government issued their white paper "Milk Policy" laying down lines for future legislation. One proposal is to empower local authorities, under certain conditions, to apply to the Minister of Health to make compulsory the efficient pasteurisation of milk sold by retail in its area. Exempted from this requirement will be milk from tuberculin-tested herds. In view of the importance of *safe* milk for the community it is to be hoped that the medical profession will give the strongest support to this wise, but long overdue, proposal.

The fact that the changes produced in milk by pasteurisation are negligible (the reduction of vitamin C not being of practical importance) has been so often demonstrated that it is unnecessary to reiterate it. I discussed these changes in detail in THE LANCET in 1933 and since that date there have been several important investigations confirming the full equality of pasteurised milk with unheated milk as regards its nutritive value.

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MEDICINE AND THE LAW

Romance

SUCCESS in their profession is for some an uphill journey. To Dr. Thomas Ronald Hyman Max Davies it came swiftly. Just as Adam, the first man, was spared all infantile complaints, so Dr. Davies escaped the tedious years of medical training and examination. Adopting the name and degrees of a medical practitioner who had died in Norfolk, he found his qualifications ready made. Owing to the misadventure of a conviction for blackmail at Winchester assizes last month, his professional career has been temporarily interrupted. For his biographical details we have only the bald statements of a detective inspector who, after the conviction and before the sentence, gave the court such information as was officially available. The record, supplemented by the enterprise of journalists, was full of romance. The birth certificate of the accused was in the name of George Maxim Davidson. He served as a seaman in the mercantile marine and was afterwards an engine-cleaner in an aircraft undertaking at Croydon. He married and had two children. Five years ago he left his family abruptly. His wife never saw him again till he stood in the dock at the Portsmouth police-court last September. He begins a new life as Dr. Davies, M.R.C.S., L.R.C.P., claiming, it is said, to have acted as locum tenens for a Harley-street consultant. He marries a lady whom he meets at a dance. His practice evidently prospers, for he lives expensively. He administers 14 injections for diabetes to a well-known theatrical artist who is later informed by another doctor that he is not suffering from that disease at all. He treats several women for large fees. He is said to have attended maternity cases, carrying the usual surgical instruments. The police suspect him, though they admit they have no evidence of this, of making money by criminal abortion. He obtains £553 from a rich widow in London apparently on the plea that he was fighting the General Medical Council. He is awarded £250 special damages in the High Court under the name of Dr. Davidson for injuries alleged to have been sustained in a motoring accident; the defendants seem to have missed a rare opportunity for an interesting cross-examination. The police say that, when arrested, he was planning another claim against an

insurance company in the name of Dr. Douglas Colquhoun for serious damage due to a traffic accident. His forensic experience was his undoing. His second wife meets at Portsmouth one of the ever-increasing tribe of "Mr. X." Dr. Davies demands money under threat of divorce proceedings. There is one trivial obstacle in his path. Being still married to his first wife, he is technically unable to divorce his second. Dr. Davies, however, does his best. He has an acquaintance called Duke, an ex-railway official who has been compensated with £1800 on retirement from a redundant post and who is now a taxi-driver. Duke is asked to assume the name of Medway and is cast (though Duke himself denies this on oath) for the part of the solicitor who is concerned with the divorce proceedings. Dr. Davies, Duke, and Mr. X meet in London, but eventually the police intervene. Dr. Davies is found guilty on ten counts of fraud charged in different forms. He pleads guilty to bigamy. There are four outstanding charges, one for forgery of a motoring licence, which the court is asked to take into account. The judge, in sending him to penal servitude for four years, seems almost to apologise for his leniency. Bad as he finds the case to be, he can imagine worse; so the maximum of five years is rightly deemed inappropriate.

Unless it was one of the unspecified charges taken into consideration in passing sentence, the offence of masquerading as a doctor, it may be observed, was not before the court at the trial. The country shows no concern over this minor aspect of Dr. Davies's career. Had he been tried under Section 40 of the Medical Act, there would doubtless have been an argument as to whether he had acted "wilfully and falsely"; his patients would doubtless have gone into the box to testify their gratitude for the benefits of his treatment; and the public would doubtless have suspected that this was just another persecution of a brilliant unorthodox practitioner by the hide-bound representatives of old-fashioned medicine. Be that as it may, it is perhaps worth noting that, almost at the moment of Dr. Davies's conviction, the architects obtained the second reading of a Bill which is to prevent anybody from calling himself an architect unless he is a "registered architect" under the Act of 1931. The Bill has none of the old nonsense of "wilfully and falsely." And the House of Commons is told that the Bill is necessary in order to give architects the same degree of protection as the medical profession enjoys. It seems not to have been appreciated that the medical profession has no more protection than the architects have already obtained under the 1931 Act. Architects are to be given what is denied to doctors. The Bill, said the proposer, was necessary for the protection of the public; laymen were defrauded if, in their ignorance, they employed an unqualified man. Architects, said the seconder, are not made in a night; it takes years of training. But why all this fuss about curricula and examinations? Whether we are legislating for those who build houses or for those who merely attend to the health of the people, the romance of Dr. Davies has shown us a far, far better way.

Delusional Insanity and the Disposing Mind

Mr. Justice Langton gave judgment on Dec. 21st in the prolonged dispute over the disposal of the £180,000 estate of the late Mr. A. H. L. Bohrmann. Two solicitors (who were the executors) propounded a will and four codicils thereto. The general effect of these documents was to give the bulk of the estate to charities in the United States, subject to a few legacies to relatives. The next of kin contested the will and codicils on the ground that Mr. Bohrmann suffered from delusions and obsessions which made him incapable of making a valid will. One of the witnesses called on behalf of the next of kin was Dr. R. D. Gillespie. He said that he found in the evidence the symptoms of mental disease. He classified the testator as a paranoid psychopath—i.e., a person not certifiable as insane and often of good intelligence but so far lacking in the balance of fundamental instincts, emotions, and sentiments common to mankind as to be disabled from the discharge of important social responsibilities. It was the mildest form of a mental disease which was constitutional and hereditary. Mr. Bohrmann had shown a morbid capacity to bear animosity. Mr. Justice Langton said that the law did not recognise any half-way house between delusional insanity and sanity. The court had been asked to say that the category of persons described as paranoid psychopaths (who fell short of delusional insanity) had not testamentary capacity. It was not within the judge's power to make such an advance; he was not able to say that a paranoid psychopath was incapable of making a will. Mr. Justice Langton was, however, satisfied that a delusion as regarded the London County Council was perfectly well established. This had caused Mr. Bohrmann to make an extraordinary declaration in the last of the codicils whereby the word "England" was deleted and the words "United States of America" substituted. Although the judge thought that the will as a whole must be upheld, he decided to delete that part which had been dictated by delusional insanity. It is doubtful whether there is any precedent for this course. The courts have been ready to delete parts of a will which have not been brought to the knowledge and approval of the testator. In this case the particular passage had not been brought to the knowledge and approval of the testator as a sane and balanced man.

UNITED STATES OF AMERICA

(FROM OUR OWN CORRESPONDENTS)

THE FUTURE OF VOLUNTARY HOSPITALS

ACCORDING to Mr. D. H. M. Pyle, president of the United Hospital Fund, who estimates the population of New York City in the year 1960 at 10,800,000, adequate hospital provisions for the expected population require a further investment of $371,000,000, a sum only slightly less than the present hospital investment. Can such a sum be raised by voluntary contributions? Revenues from paying patients have fallen off, and gifts have decreased by one-third since 1930.

The occasion for this speculation about the future of New York's hospitals was a special meeting of the trustees of the United Hospital Fund to consider a report on the hospital survey which has been conducted over the last two years under the direction of Dr. Haven Emerson, former health commissioner of the city and now professor of public health practice at Columbia University. This survey has called attention to the need for reform in certain directions. In Mr. Pyle's words:

"The survey has demonstrated the shocking lack of coördination between ourselves, hospitals under other types of control, convalescent homes, homes for the chronically ill, independent dispensaries and visiting nurse groups. It has proposed an integration

of our separate services, each with the other. It has recommended the extension of our services into the homes so that those who cannot afford private care in illness need not necessarily occupy hospital beds....

"Our system of voluntary hospitals, viewed in the aggregate and financially speaking, is virtually in liquidation.

"Many of our plants are fast wearing out, becoming obsolete; a third of our building investment, a half of our bed capacity, is in buildings over twenty years old. We have made no financial provision through depreciation reserves for the eventual replacement of old buildings but have relied on the hope, once justified by experience but now no longer a certainty, that the amounts necessary for new plants would be given us when required by a generous public. Our operating surpluses and spendable funds are depleted so that now we have little more than half the total of $80,000,000 available at the close of 1930.

"Nearly a half of our separate units have either no operating surplus or an amount so small as to not provide a safe minimum of working capital. By comparison with 1930, our revenues from paying patients are less than it seems reasonable we should expect them to be in view of our increased bed capacity. Increasingly we are dependent upon government payments for public charges. Our contribution support for current purposes has declined nearly a third since 1930. We now receive from that source nearly double the amount paid to us by the city in 1930. The hospitals have recently requested a change in the basis of payment that will increase these revenues another 50 per cent.

"And in the face of all this, it now seems clear that within the next twenty-five years, the hospital system of New York City, of which we are a part, must replace 24,300, or nearly half of its present beds and in addition provide 25,500 new beds."

The moral drawn by Mr. Pyle is that the voluntary agency must select for itself a function which it can fulfil well, within its financial limitations, and must coördinate its activities with those of governmental institutions and agencies. "Unless we put our house in order," he concludes, "circumstances perforce will require that government do it for us."

PROPOSED LEGISLATION FOR DANGEROUS DRUGS

The disaster caused by the distribution of an elixir of sulphanilamide which contained a highly toxic diethylene glycol has given a new impetus to the preparation of corrective legislation. The report from the Secretary of Agriculture on the deaths in this disaster includes the following recommendations:

1. License control of new drugs to insure that they will not be generally distributed until experimental and clinical tests have shown them to be safe for use. The definition of what constitutes a new drug should include (*a*) substances which have not been used sufficiently as drugs to become generally recognized as safe, (*b*) combinations of well known drug substances, where such combinations have not become generally recognized as safe, and (*c*) well known drug substances and drug combinations bearing label directions for higher dosage or more frequent dosage or for longer duration of use than has become generally recognized as safe. Exemption should be made for new drugs distributed to competent investigators for experimental work. A board of experts should be provided who will advise the Secretary of Agriculture on the safety of new drugs.

2. Prohibition of drugs which are dangerous to health when administered in accordance with the manufacturer's directions for use.

3. Requirement that drug labels bear appropriate directions for use and warnings against probable misuse.

4. Prohibition of secret remedies by requiring that labels disclose fully the composition of drugs. Many foreign countries now impose this requirement. Many drugs manufactured in the United States are exported to such countries under labels bearing such disclosure. The same drugs are sold to our citizens under labels that give no hint of their composition.

This, it is hoped, is one of the steps toward the introduction and passage of a suitable Food and Drug Act at the next regular session of Congress.

THE A.M.A. BROADCASTS TO THE SCHOOLS

"To the schools of America: your health" is the weekly greeting sent out over a national network of 88 broadcasting stations by the National Broadcasting Company's announcer from the company's studios in Chicago. Yesterday, through the kindness of Dr. W. W. Bauer, director of the bureau of health and public instruction of the American Medical Association, I was able to see the performance of the Association's weekly health play in the N.B.C. studio. Professional actors read the script, a professional orchestra supplies the incidental music, a specialist supplies "sound effects" as required. Yesterday the subject of the play was tuberculosis and the presentation exceedingly dramatic: the actors wore ordinary clothing but indulged in gestures and facial expression just as though they had been on the stage. At the close of the play Dr. Bauer summarised in a few minutes the practical lessons about tuberculosis which the story had brought out.

In order that teachers in junior and senior high schools may gain the maximum benefit from this ambitious programme of health education the Association has made available to them at a nominal cost a "pupil's work book and guide" containing questions for the student to answer and space for his answers; framework for tabulations of his observations and experiences and appropriate references to the literature of each subject. All this is arranged in weekly lessons each to correspond with the subject of the weekly broadcast. A series of "briefs" on these lessons is also published for the benefit of the teachers. These give suggestions for educational "projects" and further reference to books and papers.

The course of broadcast lessons begun in October will continue until the middle of June—that is for the extent of our school year. The main headings of the course, each with four sub-titles, are as follows: personal health, hygiene, diet, contagious diseases, preventing future illness, public health, health education, mothers and children, using health knowledge. The broadcasts are so timed that they may be heard in school hours whether the school is located on the east coast or on the west.

BUCHAREST

(FROM OUR OWN CORRESPONDENT)

PUBLIC HEALTH SERVICES

BEFORE the Liberal Government resigned it gave an account of its work during its four years of office. M. Costinescu, Minister of Health, when he took up his post regarded as his first duty the reorganising of the medical profession, and at the same time augmenting the State medical and auxiliary services. He raised the number of official physicians from 1700 to 2413. At every rural centre a dispensary has been established—in the last three years 549 have been

put up, so that at present there are altogether 1249 in the country, all well supplied with drugs.

During 1936 arsenic, bismuth, and mercury preparations worth 87 million lei were distributed amongst the dispensaries, marking a very important step in the campaign against syphilis. One of the most extensive social diseases is pellagra. Health measures alone have been inadequate, and M. Costinescu held that the solution of this grave problem is in the hands of the Minister of Agriculture, who ought to bring in strong measures checking the decay of maize. Another widespread disease that claims too many victims is tuberculosis, there being about 500,000 cases in Rumania. New sanatoriums have been built and the old ones enlarged; while in 1934 there were 1500 available beds, there are now 3000. A new system has been introduced where all candidates for public appointments have to be medically examined, those who are even suspected of being infected being rejected, lest they infect their associates. Energetic action has been taken also against typhoid fever. Owing to the defensive work by 40 mobile squads instead of the several thousand cases of latter years, at present there are altogether only 56 cases in the hospitals. Against malaria, which undermines the health of more than a million of the population, a still more energetic and efficacious campaign has been launched, in addition to the distribution of quinine. By draining the swamps the evil may be exterminated in its root, but the enormous work this involves will not be finished for about 15 years. In the four years 32 fever hospitals have been built and 40 repaired and reopened, and 40 county hospitals have been fitted with modern equipment; each has now a motor ambulance.

SHORTENED POST-OPERATIVE CONVALESCENCE

Prof. Liviu Campeanu, director of the Marzescu Hospital in Brasov, began to investigate some years ago whether it was possible safely to shorten the time of convalescence of patients after operations. He came to the conclusion that convalescence in many cases is too long. He has a series of patients who got up two days after an operation for appendicitis, and even several who stepped down from the operating table and walked to their bed. His investigations embraced about 1000 surgical patients, and the shortened convalescence did not cause any harm; in fact they were glad to be able to return to work so early. Prof. Campeanu's ideas have spread all over the world, and lately he received three distinguished foreign visitors, John H. Gordon of Boston, U.S.A., Lecutiu Traian of Detroit, and Martin Tezdal of Oslo. They were surprised, on asking patients walking about dressed as healthy people when they were operated upon, to be told by one that he underwent an operation for appendicitis the day before, and by another, a woman, for umbilical hernia three days before. Prof. Campeanu has received an invitation from America to lecture there on his methods.

NAME PLATES AFTER MOVING HOUSE

Recently a Bucharest physician moved away from his old flat and he left behind his name plate with an indication of his new address. The landlord ordered the plate to be removed, and he was summoned by the doctor at the county court. The verdict obliged the landlord to replace it on the grounds that it had become a general custom for a professional man to leave his name plate behind for a certain time. This holds even if the doctor was evicted by the landlord for non-payment of rent, or if there was a difference between them in connexion with medical fees. On the other hand, the plate can do the landlord no harm at all; on the contrary, it is an advantage to him by saving his porter the trouble of telling inquirers where the doctor lives.

A NEW PÆDIATRIC JOURNAL

Under the title of *Revista de Pediatrie si Puericultura* a new journal has been created by Prof. Gheorghe Popovici, who holds the chair of pædiatrics at the University of Cluj. Prof. Popovici collaborated in valuable research on vitamins with Prof. Paul György, formerly of Heidelberg, and now at the University of Cleveland, U.S.A. The new journal appears monthly, in a very attractive cover, and is published and edited by the Society of Pediatrics and Puericulture, Cluj.

A PRIZE FOR PSYCHIATRISTS

On his retirement, Prof. Joan Nanu-Muscel deposited at the National Bank of Rumania 800,000 leis to the credit of the Medical Academy. This will enable the award of 72,000 leis every three years to the author of the best treatise on the treatment of schizophrenia. The prize will bear the donor's name.

DEATH OF PROF. A. OBREGIA

The death is announced here of Alexandru Obregia, for 40 years professor of psychiatry in the university, from septicæmia in his 77th year. As a student he was pre-eminent, winning several scholarships, and being elected president of the Association of Medical Students. He has been regarded as the parent of cisternal puncture, now so much in vogue. His accounts of his investigations into the manic-depressive psychoses and general paralysis of the insane are known world-wide, and have been translated into several western languages. His creative genius did much in modernising Rumanian public health, and he was twice leader of the national public health administration as ministerial secretary. He founded the Central Hospital for Insanity and for Nervous Disease, and the Socola Hospital in Jassy, capital of the province of Moldowa. It was a great distinction when in 1922 during the Averescu régime, he was honoured with the position of vice-president of the Senate. The *Romania Medicala*, the leading medical journal of the country, has devoted an entire issue to his memory.

Panel and Contract Practice

The Payment of Anæsthetists

IT is seldom that the Government auditor makes a "reservation" about the accounts of an insurance committee. That the Kent committee have recently been the victims of such action need cause no disquiet, because the expenditure questioned on this occasion has been sanctioned in retrospect by the Minister under the powers conferred upon him by the 1936 Act (Section 117 (2) and the Fourth Schedule). The trouble is about the inclusion among the charges to the practitioners' fund 1936 of the sum of £118 13*s.* in respect of payments to certain practitioners assisting at operations for the services of a colleague as anæsthetist, the operation being performed by a third practitioner. The auditor held that there was no lawful authority for such payments.

According to Clause 9 (7) of the terms of service a practitioner is responsible for providing the services of an anæsthetist for any operation (other than an obstetric operation) that he is performing himself provided that the operation is of a kind usually undertaken by a practitioner, and that the administration of the anæsthetic does not involve special skill or experience. Clause 2 of the distribution scheme shows how special payment for the services of an anæsthetist may be secured (as a first charge against the practitioners' fund). When this question was considered in 1925 by the Kent panel committee, they had before them a list of cases where, in the opinion of the Insurance Acts Committee, an anæsthetic fee could be paid, and noted that these did not include cases in which the practitioner summoning the anæsthetist was himself only acting as an assistant. But the panel committee took the view that the intention of the Minister *was* to provide an anæsthetist under these circumstances and to pay for his services and that the limitations suggested by the I.A.C. followed too closely the strict legal interpretation of the regulations. The committee considered it unlikely that the Minister would raise any objection to a more liberal interpretation, as the payment would be made from the local practitioners' fund and would entail no extra cost to the central funds. Actually no official objection was raised until the accounts were audited at the end of 1936. The Minister upheld the objection then made and such payments will not be allowed in future.

The position is that a practitioner may be paid for the services of an anæsthetist only when he is required to provide those services, and this is only when he performs the operation himself. He is however bound to give either assistance or an anæsthetic as part of his obligations, without cost to the insured person. The executive committee of the panel committee have decided not to contest the decision as regards the future, but in view of the period during which claims have been met without question the Minister is to be asked to sanction payment of a number of claims made during 1937, as he has already done for those made during 1936.

PUBLIC HEALTH

Confidential Reports

AT a recent meeting of Caernarvonshire combined sanitary committee a protest was made against a refusal of the Minister of Health to supply Gwyrfai council with a copy of a report on the district by one of H.M. inspectors. The clerk observed that the question whether reports made by the Ministry of Health inspectors should or should not be submitted to the authorities concerned had long been "a bone of contention." Surely there is some misunderstanding here. These inspectors are the servants of the government departments to which they are attached and their investigations are submitted as confidential communications. It lies with the Minister concerned to decide what shall be done with them. If he sees fit, and often he does, he issues a communication to the authority concerned, but he could hardly be expected to supply on demand his official correspondence. A more common complaint by a sanitary authority is that it does not see the annual report of its medical officer before it is published and copies have been sent to the Minister of Health. Here the position is slightly different, for the medical officer of health owes allegiance to the local authority that appoints him and pays his salary. But in fact the Ministry of Health mostly pays directly or indirectly half this salary, and except in a very few places has to give consent to the appointment or dismissal of a medical officer. In return he claims the right to see the annual reports before they are presented to the local authority. In the few boroughs where the Ministry has no control of this kind, the annual report need not go to the Ministry before it is passed by the local authority. It is a grievance of local authorities that their medical officers may say something which is distasteful to them and that they have no power to prevent its publication. But this privilege lies at the very foundation of our system of public health administration. It is very seldom abused and if it is the local authority has a remedy. For though the medical officer cannot be dismissed without the consent of the Minister of Health, this consent would only be withheld if the officer is likely to be victimised for doing his duty. Every year some authorities register objections to the annual reports, but as the objections are usually to unpleasant truths, nothing comes of them. We have not till now heard of any local authority openly objecting to the refusal of permission to see the reports of the Ministry's inspectors, though we are well aware that many would like to see and edit them before they reach Whitehall.

Youthful Offenders

The Home Secretary has sent a circular letter to the clerks of justices suggesting that juvenile offenders should sometimes be placed in lodgings or in a family in place of being sent to an institution. The courts already have power to provide for the maintenance of offenders under 21 in approved hostels from which they can go out to daily work, and the Government is now prepared to assist financially a much wider use of this method. An unruly child of 14 or 15 may be better in an approved home than in an approved school, and in future a grant may be made from public funds for this purpose also.

A memorandum sent to the Home Secretary by the Parliamentary penal reform group urges him to use the proposed observation centres as clinics. This group is convinced that not only personal suffering but cost to the community would be saved by psychiatric treatment in suitable cases, and it would like the Home Secretary to make further use of the powers he possesses of trying out measures of penal reform. Happily Sir Samuel Hoare has already shown himself inclined to make such experiments.

A List of Sanatoria

A new edition has been issued by the Ministry of Health of the list of sanatoria and other residential institutions approved for the treatment of tuberculous persons in England and Wales. Most local authorities are obliged to rent such beds from the voluntary bodies during the summer months; some, such as the L.C.C., do so all the year round. The list has its value in selecting institutions for this purpose. The Ministry's approval implies a certain standard and is to that extent a check on the type of institution available. The list (10*f*) can be had from H.M. Stationery Office for 6*d*.

GRAINS AND SCRUPLES

Under this heading appear week by week the unfettered thoughts of doctors in various occupations. Each contributor is responsible for the section for a month; his name can be seen later in the half-yearly index

FROM A BACTERIOLOGICAL BACK-NUMBER

I

WHEN Jenner and Pasteur developed the idea of artificial immunisation they did something more than make a scientific discovery; they founded a faith and as so often happens with faith came an offset of superstition and charlatanry. Neither of these great innovators approached the matter as entirely unprejudiced and impersonal observers. They aspired to be missionaries as well as scientists. It seems odd in our disillusioned age that the nineteenth century should have found its sermons so readily in stones though it was perhaps the gift for identifying what *is* with what *should be* that made it both great and ridiculous. Immunisation was born and brought up in rose-coloured surroundings and it has continued to live in a dangerous atmosphere where the wish has been wont to be the father of the thought.

* * *

We have lived through some striking changes in the attitude to small-pox vaccination. Compulsory vaccination which once had the suffrage of the nation has now hardly a serious supporter. We are ashamed to jettison the idea completely and perhaps afraid that if we did the accident of some future epidemic might put us in the wrong. We prefer to let compulsory vaccination die a natural death and are relieved that the general public is not curious enough to demand an inquest. In the meantime our attention is diverted to other and newer forms of immunisation. In our own immediate times diphtheria immunisation has taken its turn as the arch stunt of the immuniser. The scientific ideas behind it were certainly engaging and were proved beyond any reasonable doubt. The Schick test distinguished the susceptible from the immune and by a simple treatment the susceptible could be made immune. These facts carried with them no categorical imperative. That diphtheria can be prevented by immunisation no more implies a command to immunise people than the fact that nitric acid and glycerin make an explosive mixture implies a command to blow up our neighbours. Yet the immunisation of the masses has been undertaken with almost a religious fervour. The enthusiast rarely stopped to wonder where it would all finish or whether the fulsome promises made to the public in the form of "propaganda" would ever be honoured. Without propaganda there can, of course, be no large-scale immunisation, but how perilous it is to mix up propaganda with scientific fact. If we baldly told the whole truth it is doubtful whether the public would submit to immunisation. On the whole diphtheria immunisation has proved a fairly safe affair, but suppose we included in our propaganda a candid account of the various untoward accidents which have accompanied the procedure. No method involving a parenteral injection is without a significant risk. When injecting a healthy individual with anything we are always skating on thin ice. Sick people for the most part are quite prepared to take a risk in trying out a remedy, but the main desire of well people is to preserve their status quo. If you knock them out in an effort to protect them from a disease there is no knowing they will ever get, there is the devil to pay. Accidents and mistakes must inevitably happen and when they take place what might have been a highly instructive lesson is usually suppressed or distorted out of recognition. Those who have had to take detailed notice of the immunisation accidents of the past few years know that to get the truth of what really went wrong generally calls for the resources of something like a secret service. And if the technical experts have often been furtive and disingenuous in their methods the public in its turn is unfair and vindictive. The whole world is ready to be wise after the event and a scapegoat has to be found at all costs. Some of the mistakes that have been made seem foolish enough when coldly reviewed: mislabelled bottles, preparations issued without check tests on animals, the omission of a disinfectant—all this seems easily avoided but the point is that they *are* made and are bound to be made sometime or other, even with the greatest vigilance. There are few immunologists responsible for the preparation of immunological reagents or doctors who have carried out immunisation on a large scale who have not had some hair-raising experience.

* * *

My own most unhappy experience in this direction was when immunising a group of mentally defective children against scarlet fever. It was in the early days of scarlet fever immunisation but we had already had a reassuring experience with many hundred children. Of course we knew that we had to deal with a population that might contain some pleasant "unknowns" and we ran a preliminary trial on two or three dozen of the children to see how things would go. Everything went well, so as time and the occasion pressed, we proceeded to immunise the rest of the children. We had not got very far when we were urgently called back to the bed of one of the injected children and there found a Mongol imbecile pulseless and looking like death. In the next few minutes came four more summonses of the same kind and each of the unfortunate patients was a Mongol imbecile. We had failed to include a specimen of Mongol in our try-out—a stupid enough thing to do, for as everyone knows Mongols are odd little folk and a law unto themselves. All's well that ends well and fortunately all the patients recovered with almost the same speed and inconsequence with which they had collapsed. Even to this day we don't know the reason for this strange behaviour of Mongols and needless to say we were deterred from further experiments on such kittlecattle. We may not have learnt anything of the essential nature of mongolism but we *did* learn to think more feelingly of the misfortunes of our fellow immunisers.

The risk of accidents must remain one of the snags of all forms of immunisation. The individual immuniser is after all the only person who has the moral right to decide whether the game is worth the candle. His knowledge may be quite inadequate for the decision but the risks are one way or another mainly personal and his concern alone.

* * *

The gradual extension of immunisation to an increasing number of diseases is rapidly bringing us

to the stage when we have too much to choose from. Shall I be immunised against small-pox, diphtheria, and typhoid (a standard selection), or shall I choose influenza (by the new method), the common cold (by the old method), and scarlet fever? Shall I be high-brow and fortify myself against psittacosis and tularæmia or prepare myself for a really *grand tour* by having a few shots against the fevers of Malta, the Rift Valley, or the Rocky Mountains? It is quite clear that if I don't want to tattoo myself from head to foot with injections or come out in as many rashes as a chameleon I have got to stop somewhere. My risks of contracting even the commonest of these diseases is usually small—not nearly so great as my risk in crossing the High-street between any Sainsbury's and any Woolworth's.

Can we ever abolish a human disease by a mass immunisation? While the craze is hot we can immunise a thousand or ten thousand, but sooner or later the apostolic zeal wanes, or the available hoardings are wanted for some more fashionable advertisement and the Sisyphean stone once more rolls down the hill and we are much in the position as we were at the beginning. Immunisation surely should remain a matter of private, not of public, venture—a question for the individual to decide on personal grounds and in terms of his own risks, fears, and prejudices.

We see the same impasse reached in the current argument about typhoid inoculation. Sir John Ledingham rightly laughs at the risks of the negative phase. As a distinguished bacteriologist noblesse oblige that he should not be pusillanimous in the exercise of the special methods of his own subject. Your LANCET critic is inclined to pooh-pooh the risks of disease and dwell on the risks of inoculation. He seems to prefer the God-sent to the man-sent risk and perhaps in this he is also right. The Mayor of Croydon is said to have discussed the typhoid epidemic over a dish of watercress. Perhaps he is rightest of all. After all if the chicken had listened to other chickens it would never have dared to cross the road, and it would never have got to the other side—Q.E.F.

PARLIAMENTARY INTELLIGENCE

RISE IN THE COST OF LIVING

IN the House of Commons on Dec. 22nd Mr. LATHAN moved:

"That this House, taking note of the upward trend of prices without a corresponding increase in the income of the average household, is of opinion that the public, especially that section of it already suffering on account of inadequate resources, should be protected by measures for the better organisation of production and distribution and the elimination of profiteering in order to keep the cost of living within proper limits."

He said that in the last four years food prices had risen 16 per cent. Milk was dearer to-day than in any other country in Europe. The desirable amount of milk for children was from one to two pints a day; at present prices 1½ pints a day would cost 3*s.* 4½*d.* a week, more than the sum allowed by the Unemployment Assistance Board for a child under five. In 1933 the cost of a minimum diet was said to be 22*s.* 6½*d.* per week; it had now risen to from 26*s.* 8½*d.* to 36*s.* 2*d.*—figures obtained in the cheapest shops doing ordinary working-class trade.—Mr. RIDLEY, seconding the motion, said that the Government policy was largely to blame for the rise in food prices. The country had gone back in the last five years to economic nationalism.

Mr. RAIKES said that he knew of no better wicket upon which the Government could bat than upon the rising cost of living at present. A fall in the price level would mean a gradual return to the conditions from which the country had drawn itself during the last few years. Cheap talk about the cost of living obscured the fact that to-day the purchasing power of the nation was higher than at any previous time.—Captain MACNAMARA pointed out that the cost of living was considerably higher in France, Germany, Italy, and the United States.

After further debate, Captain EUAN WALLACE, Parliamentary Secretary to the Board of Trade, said that the recent rise in the cost of main foodstuffs was mainly due to the widespread strengthening of demand caused by the general economic revival. During the depression a rise in prices was declared by the leaders of all parties to be an essential condition of recovery. Those who had been in constant employment throughout the depression might be slightly worse off now than four years ago; but there was no doubt that to-day the insured population as a whole was better off. The cost of living index was lower than in 1929, the last year of prosperity, and wages were higher; consumption of food, drink, and tobacco was 20 per cent. greater; the total expenditure on social services had risen from some £468,000,000 in 1930, when the Labour party were in office, to over £503,000,000 in 1935, the last year for which figures were available; to-day 3,000,000 children were receiving cheap and 400,000 free milk; when Labour held office 27,500,000 free meals were given to children by local education authorities, whereas now there were 100,000,000; the sale of milk had gone up by 16,000,000 gallons during the last year. Special attention was being paid to households where a substantial part of the income came from the Unemployment Assistance Board. The first review of cases was completed on Nov. 30th and increased allowances were being made in some 250,000 cases. It would be impossible to run a contributory pensions scheme on the basis that payments should fluctuate with the cost of living; if that were done the 10*s.* old age pension would be worth 6*s.* 9*d.* to-day.

The motion was negatived.

NOTES ON CURRENT TOPICS

The Christmas Adjournment

The House of Lords adjourned for the Christmas recess on Dec. 22nd, and the House of Commons on Dec. 23rd. Both Houses, unless called together earlier in emergency, will reassemble on Feb. 1st.

Coal Bill and Miners' Welfare

On Dec. 20th, when the House of Commons considered the Coal Bill in Committee, a series of amendments which were all negatived were moved to provide for miners certain benefits in the allocation of the surplus. The suggestions included a pension fund for those over 55, funds for improving their conditions, especially in regard to safety, noise, and silicosis, and for displaced miners in general.

Mr. OLIVER STANLEY, President of the Board of Trade, did not think that any contribution that a Commission of this sort could make year by year as its surplus rose or fell, or disappeared, would be in any way a safe foundation upon which a pension scheme, however desirable in itself, could possibly be based. The question of safety must be left for decision by the Royal Commission that was considering this and other matters. Already a large proportion of the cost of research would be borne by the Coal Commission. If work of this sort was being starved for money—and he did not think that there was any ground for saying that—it was surely not right that there should be delay by waiting for the surplus fund to provide a remedy.

QUESTION TIME

TUESDAY, DEC. 21ST

Compulsory Vaccination at Woolwich Arsenal

Mr. LEACH asked the Secretary of State for War whether he was aware that Cyril Francis Youngman, of 350, Woolwich-road, Charlton, aged 14 years, died from encephalitis resulting from vaccination on Dec. 2nd, the vaccination having been undergone so that the lad could take up a situation at Woolwich Arsenal; that the Minister of Health, in his recent annual reports, had recommended that children of school age and young adults should not be primarily vaccinated unless in special danger of smallpox, as this operation might cause or activate encephalitis; and whether, in view of this case, he would abolish the regulation requiring certain workers at Woolwich Arsenal to be vaccinated.—Mr. HORE-BELISHA replied: I have been made aware of the unfortunate circumstances in which this lad died, and would like to express my sincere sympathy with his family. Such cases are very rare, but I am considering whether the present regulation requiring the vaccination of entrants to the Royal Arsenal should be relaxed.

Prevention of Silicosis

Mr. JAMES GRIFFITHS asked the Chancellor of the Duchy of Lancaster, as representing the Lord President of the Council, if he was aware that an investigation had been conducted by a group of scientists at the McIntyre mine, Ontario, Canada, into the possibility of the prevention of silicosis by the admixture of metallic aluminium with silica dust; and, in view of the claims made that the initial experiments revealed possibilities of successful prevention, if he would undertake to have these experiments studied by his expert advisers and consider the possibility of conducting similar experiments in this country.—Earl WINTERTON replied: The progress of these Canadian investigations is being followed with interest by the special committee advising the Medical Research Council in this subject, and some experiments on similar lines have already been initiated in this country.

Salaried Midwives

Sir ROBERT YOUNG asked the Minister of Health the number of salaried midwives appointed since August 1st, 1937; what was their distribution throughout the counties of England; and whether there had been any difficulty in getting the requisite number required in any part of the country.—Mr. BERNAYS, Parliamentary Secretary to the Ministry of Health, replied: The number of salaried midwives included in the proposals of local authorities, under the Midwives Act, 1936, is approximately 7550, and I am sending the hon. Member particulars of the geographical distribution of this number. I think it is safe to assume that a large majority of the midwives have already been appointed. Returns showing the exact numbers appointed by the end of this year will be available in February next. As regards the last part of the question, my right hon. friend is not aware that any serious difficulty has been experienced in any part of the country in securing the requisite number of midwives.

WEDNESDAY, DEC. 22ND

Asbestosis Certificates

Mr. KELLY asked the Home Secretary if he was aware that Mr. Edmund Ashworth, late of 10, Abbey-street, Rochdale, died on August 17th last of asbestosis, but that the medical board had not issued their certificate in spite of repeated efforts; and would he take steps to expedite the issue of this certificate and to prevent future delay in issuing asbestosis certificates.—Mr. GEOFFREY LLOYD, Under Secretary, Home Office, replied: I understand that exceptional difficulties arose in this case because no application to the Board was made until the middle of September, and when the Board endeavoured to collect the evidence as to the cause of death, it was found that practically none of the material from the post-mortem was still available. The Board reached a decision early in November but subsequently the solicitor who had made the application informed the Board that the widow had died and that a certificate was no longer required. It has been impressed upon the Board that there should be no avoidable delay, but their difficulties are much increased if applications for certificates in fatal cases are not made immediately on the workman's death.

Roller Towels and Infection in Factories

Mr. TURTON asked the Home Secretary whether, in view of the risk of infection attached to the use of roller towels, he would make regulations under Section 42 (2) of the Factories Act, 1937, providing that the provision of roller towels would not be deemed adequate and suitable washing facilities.—Mr. GEOFFREY LLOYD, Under Secretary, Home Office, replied: Roller towels are use in a large number of factories, and my right hon. friend has no evidence at present which would justify him in taking the line that they are generally unsuitable on account of risk of infection. He will however have further inquiry made into the point.

Sleeping-sickness Research: Native's Bravery

Dr. HADEN GUEST asked the Secretary of State for the Colonies whether, in connexion with the recently published annual report of the Colonial Development Advisory Committee, he would ask for the name of the Tanganyika African who was mentioned as having allowed himself, in the course of investigations into the transmissibility of sleeping-sickness, to be infected with the disease, in order that his service to the cause of science and public health might be recognised.—Mr. ORMSBY-GORE replied: The name of the volunteer in question was Ngasa. I should like to take this opportunity of expressing my appreciation of the bravery which has been shown by this man, and others connected with this sleeping-sickness research, in exposing themselves to infection. Ngasa made a most satisfactory recovery.

Health Insurance and Women's Benefits

Mr. BUCHANAN asked the Minister of Health when it was proposed to restore the cuts made in national health insurance benefits to women which were reduced by the Act of 1931, particularly with regard to the improvement in the financial position of the funds and also seeing that almost every other cut made had been restored.—Mr. BERNAYS, Parliamentary Secretary to the Ministry of Health, replied: The reductions to which the hon. Member refers were not due to the needs of national economy but to the increased claims for benefit in respect of women by which the solvency of the scheme was endangered. The number of claims has not fallen to an extent sufficient to enable the question of increased benefits to be considered.

Tuberculosis Services in Wales and Monmouth

Mr. JENKINS asked the Minister of Health when the report of the committee of inquiry into the anti-tuberculosis services in Wales and Monmouthshire would be completed; and whether it was intended to publish the report.—Sir KINGSLEY WOOD replied: No, Sir. I understand that the various bodies who wish to give evidence before the committee are being asked to complete and submit a written summary of their evidence before the end of the year, and the committee hope to hold a public sitting in Cardiff early in the New Year. Further sittings will be necessary before the inquiry can be closed. As regards the second part of the question, I will consider the question of publication of the report as soon as I receive it.

THURSDAY, DEC. 23RD

Leprosy in this Country

Mr. MESSER asked the Minister of Health if he was aware that there was only one small colony for 12 patients suffering from leprosy in this country, and that a Middlesex patient of high infectivity could not be admitted recently owing to lack of accommodation; and if he would sanction financial support to this colony to enable it to be extended to admit all patients deemed to be infective in character.—Sir KINGSLEY WOOD replied: I am aware of the circumstances to which the hon. Member refers. I am advised, however, that prolonged and close contact with an infected

person is necessary for the transmission of this disease and that in the conditions normally existing in this country the risk of transmission is negligible. I understand further that the patient in question is now in a London hospital and that she will be admitted to the colony as soon as a bed is available. The local authority in whose area she resides are prepared to make a substantial contribution to her maintenance and steps are being taken by the managers of the colony to raise voluntary funds for an extension of the accommodation.

Mr. MESSER asked the Minister of Health how many cases of leprosy in this country had been reported to the Ministry of Health; how many were known to have been contracted here; and how many were regarded as being of high infectivity.—Sir KINGSLEY WOOD replied: Cases of leprosy are not required to be reported to my department, but I have information of 38 such cases which have come to their notice in this country. I know of only 4 cases over a long period of years in which it is considered that the infection was incurred in this country. In each of these there was a history of prolonged and intimate contact with a person who contracted the disease abroad. I have no information with regard to the last part of the question.

Typhoid on s.s. *Strathaird*

Mr. JAMES HALL asked the Minister of Health if he was aware that there was an outbreak of typhoid on board the s.s. *Strathaird* on her last homeward voyage in October; whether any passengers suffering from typhoid were landed at Marseilles; whether the members of the crew were inoculated against typhoid; whether the passengers and crew landed in London who were possible contacts were followed up; and whether or not any of them were traced to Croydon.—Sir KINGSLEY WOOD replied: I am aware of the outbreak to which the hon. Member refers. From the information in my possession it does not appear that any passengers suffering from typhoid were landed at Marseilles. I understand that the members of the crew were not inoculated. The names and addresses of the 329 passengers and of the 233 European members of the crew were sent by the London Port Health Authority to the medical officers of health of the districts of destination. Three passengers and two members of the crew gave addresses in Croydon, and I am informed by the local authority that they were followed up and are reported to have remained in good health.

Mental Hospital Accommodation in Lancashire

Mr. KELLY asked the Minister of Health whether steps were being taken to deal with the shortage of accommodation in Lancashire for mental patients.—Sir KINGSLEY WOOD replied: Yes, Sir; steps are being taken to extend the existing mental hospitals and the Brockhall mental deficiency colony, and to provide a new hospital and a new colony.

Disease Carriers

Mr. GROVES asked the Minister of Health whether, amongst the notifications of disease sent to his department, so-called carriers of disease were included; and whether these carriers were isolated in infectious diseases hospitals or anywhere else.—Sir KINGSLEY WOOD replied: No, Sir. The statutory obligation to send notice of disease to the medical officer of health of the district concerned relates only to persons suffering from the disease and it is only such persons who are appropriate for admission to an isolation hospital. Persons who are known to be potential sources of infection but do not show any clinical symptoms of disease should be normally the subject of appropriate precautionary measures at home.

Health Insurance and Ophthalmic Benefit

Mr. HUTCHINSON asked the Minister of Health how many approved societies gave additional benefit for ophthalmic treatment to all classes of their members; and whether, in the case of societies which did not give ophthalmic benefit to all classes of their members, he would take steps to ensure that ophthalmic benefit, or some modified scale of ophthalmic benefit, should be given to those members not at present entitled to benefit.—Sir KINGSLEY WOOD replied: Ophthalmic benefit is included amongst the additional benefits available to the members of 730 approved societies and 4700 branches, with a total membership of 11,300,000. Members of these societies only become entitled to the benefit after a waiting period of $2\frac{1}{2}$ years on average, but after the completion of the waiting period the benefit is available to all classes of members. Legislation would be necessary to make ophthalmic benefit available to all members of all approved societies, and such legislation cannot at present be contemplated.

Local Government and Public Health in Scotland

Mr. MATHERS asked the Secretary of State for Scotland whether he was now in a position to make any statement as to the prospect of consolidating the Local Government and Public Health Statutes relating to Scotland.—Mr. ELLIOT replied: Yes, Sir. After consultation with the local authority associations I have decided to appoint a representative committee to review the whole field of local government and public health law in Scotland and to prepare the necessary measures for its revision and consolidation. The committee will comprise representatives of the political parties, of the local authority associations, and of the Government departments concerned. Sir John Jeffrey has agreed to act as chairman of the committee. The constitution of the committee is as follows: Mr. George Cruickshank; Mr. Matthew G. Fisher; Sir Robert Hamilton, M.A., F.R.G.S., F.S.A. (Scot.); Mr. William Hutton, M.A., LL.B.; Sir John Jeffrey, K.C.B., C.B.E.; Mr. J. Graham Kerr, M.P.; Mr. J. L. Mackenzie, M.A., LL.B., J.P.; Mr. Norman S. Main; Mr. David Milne; Mr. David Robertson, LL.B.; Mr. P. J. G. Rose, C.B.; Mr. J. M. Vallance, W.S.; Mr. R. Bryce Walker, M.A., LL.B.; and Mr. Joseph Westwood, M.P.

The following are the terms of reference: "With a view to the consolidation of the enactments applying to Scotland and dealing with (*a*) local authorities and local government and (*b*) matters relating to the public health, to consider under what heads these enactments should be grouped in consolidating legislation and what amendments of the existing law are desirable for facilitating consolidation and securing simplicity, uniformity and conciseness."

Alleged Assault by Warders on a Prisoner

In a reply to Mr. MCGOVERN concerning injuries received by Francis Healy at Barlinnie Prison on Sept. 10th, 1937, Mr. WALTER ELLIOT, Secretary of State for Scotland, said he was satisfied that no undue force was used by the warders. "Consideration of the course of events in the present case, however, has shown the desirability of ensuring that, if a prisoner whose sanity is in doubt becomes violent, there should be present from the earliest possible moment the medical officer, or, in his absence, the prison governor. Directions, as I informed the House on Nov. 23rd, have been given accordingly."

Smoke Abatement in London

Sir PERCY HARRIS asked the Minister of Health how many prosecutions there had been in London during the last 12 months under the Public Health (London) Act, 1891, and the Public Health Smoke Abatement Act, 1926, for chimneys sending forth smoke in such quantities as to be a nuisance; whether there were any officers of his department detailed to see that the provisions of these Acts relating to smoke abatement in London were enforced; and whether he was satisfied that all the London boroughs were making full use of the powers entrusted to them.—Sir KINGSLEY WOOD replied: The latest information I have is for the year 1936 when two prosecutions were reported. The answer to the second part of the question is in the negative. The statutory responsibility for enforcing the smoke abatement law in London rests on the metropolitan borough councils. The London County Council have certain default powers and also powers of making complaint to me. I have received no complaint of failure by the councils to use their powers.

CORRESPONDENCE

CARCINOMA OF THE STOMACH

To the Editor of THE LANCET

SIR,—I hope that the correspondence on this subject will not come to an end before definite steps are taken to settle the question in dispute.

I write as an ignoramus, but I am very anxious to know whether cancer is often a sequel of peptic ulcer. It is said that cancer can follow chronic gastritis. It is also said that peptic ulcer may originate in inflammation. Perhaps there is more support for the second statement than the first.

But does a peptic ulcer often become cancerous? Who shall answer this question and on what grounds? The surgeon may be able to diagnose a cancer at operation but he will have difficulty in saying whether it started from a peptic ulcer. He sometimes cannot be certain whether an ulcer is cancerous or not. The radiologist can give an opinion but he is not likely to be dogmatic about doubtful cases. The gastroscopist may recognise the nature of an obvious cancer, but he is not likely to be able to give an opinion with certainty about the less obvious. The morbid anatomist has the best chance and his opinion is the one that should be most seriously considered. There are certain criteria which the morbid anatomist can apply and on the basis of his opinion trustworthy statistics would be built. In his capacity as a histologist he would be able to gather further evidence. But it is to be remembered that on this question even histologists can err.

No amount of theorising nor quoting of authorities is going to settle the question. Let us have the matter thoroughly investigated by physicians, surgeons, radiologists and above all by morbid anatomists of our teaching schools. Let them take time so that they can produce statements that cannot be disputed.

I am, Sir, yours faithfully,

Portland-place, W., Dec. 21st, 1937. CHARLES MILLER.

TREATMENT OF SHOCK

To the Editor of THE LANCET

SIR,—I was much interested in Dr. Walker Tomb's article on shock in your issue of Dec. 18th, having myself put forward similar arguments in favour of the sympathetic theory (*Bgham med. Rev.* 1936, **11**, 131; *Lancet*, 1935, **2**, 1412; *Brit. med. J.* 1935, **2**, 1227). I think it might be dangerous, however, to pass unchallenged the view suggested that spinal anæsthesia is always a good treatment for shock. There is no doubt that under the influence of a spinal anæsthetic the signs of shock will sometimes vanish, but in others they are made worse. I believe the explanation is as follows: In early cases where the amount of plasma lost from the circulation is relatively small, cutting off the painful impulses does good, and there is enough blood in currency in spite of the loss of tone produced in the vessels of the lower part of the body. Where, however, the plasma-loss is more severe the vasoconstriction present is absolutely necessary to carry on the circulation, and it can only be overcome gradually if the patient's life is not to be endangered. In such a case the sudden paralysis of a large part of the sympathetic system increases the collapse and may produce death.

Even in the prevention of shock the protection of spinal anæsthesia is not complete. If we accept the evidence of a rising blood-sugar, sympathetic hyperactivity goes on even in an analgesia to the nipple plane, which means that all sympathetic impulses to the liver and adrenal glands are not cut off, contrary to the teaching of the late Howard Jones. In the cat sympathetic fibres to the splanchnic area have been shown to emerge from as high as the second thoracic nerves. Local reflexes confined to the splanchnic area may also play a part in abdominal operations but these can be stopped by splanchnic block. In "high spinal," therefore, anxiety on the part of the patient will cause a continued secretion of adrenaline, and will also cause sympathetic impulses to be conveyed to those parts of the body unaffected by the analgesic. The patient's anxiety can be reduced by a basal hypnotic, and its effects can be stopped completely by the use of gas-and-oxygen, which apparently has an inhibitory effect on the sympathetic system, as no rise in blood-sugar occurs.

Dr. Tomb's suggestion of treatment with ergotoxine is logical, though like him I have not tried it. A simple method of treatment, however, is to give insulin, which is antagonistic to the sympathetic, in small doses repeated as often as required and controlled if necessary by blood-sugar estimations. Fluids of course should be given to replace loss by sweating and otherwise.

I am, Sir, yours faithfully,

J. W. RIDDOCH.

Hagley-road, Birmingham, Dec. 24th, 1937.

APLASTIC AND ACHRESTIC ANÆMIAS

To the Editor of THE LANCET

SIR,—In your issue of Dec. 11th, Dr. A. F. Zanaty describes the results of sternal puncture in a number of cases which he calls achrestic anæmia; from his findings and certain other points he arrives at the conclusion that achrestic anæmia is a variety of pseudo-aplastic anæmia, and disagrees with our suggestion that it may be related to pernicious anæmia. But Dr. Zanaty's cases present many differences from our published description of achrestic anæmia. The points he quotes in support of his theory are that the cases he examined showed: (1) a macroblastic hyperplasia of the bone-marrow; (2) a marrow less cellular than in pernicious anæmia with many nucleated red cells in small groups; (3) more or less hypoplastic white-cell elements; (4) anisocytosis of the red cells "rarely extreme"; (5) a higher reticulocyte count than in pernicious anæmia; (6) the occasional presence of immature leucocytes; (7) usually a negative van den Bergh reaction; (8) occurrence at a *younger* age than in pernicious anæmia; (9) absence of nervous complications; and (10) a remarkable tendency to hæmorrhage.

Taking these points seriatim, the cases quoted in our final description of achrestic anæmia [1] showed: (1) a type of hyperplasia of the bone-marrow resembling that of pernicious anæmia—i.e., megaloblastic; (2) marrow equally, if not more, cellular than in pernicious anæmia with groups of hæmocytoblasts (megaloblasts) the prominent feature; (3) white-cell elements sometimes hyperplastic and sometimes hypoplastic, as in pernicious anæmia (see Muir [2]); (4) anisocytosis severe when the anæmia was severe; (5) a low reticulocyte count, in the absence of intensive liver therapy—as in pernicious anæmia; (6) immature leucocytes occur occasionally in typical pernicious anæmia; none of our cases of achrestic anæmia happened to show any; (7) the van den Bergh reaction varied from negative to indirect or direct

[1] Israëls, M. C. G., and Wilkinson, J. F., *Quart. J. Med.* 1936, n.s. **5**, 69.
[2] Muir, R., Text-book of Pathology, 3rd ed., Edinburgh, 1933.

delayed—as in pernicious anæmia; (8) we have exceptionally seen pernicious anæmia in a girl of 19; three of our cases of achrestic anæmia were over 60; (9) the patients may not have survived long enough to show nervous complications, which are notable only in a proportion of true pernicious anæmia patients; (10) none of our cases had any hæmorrhage, in fact when discussing the differential diagnosis we state that the presence of hæmorrhage suggested aplastic anæmia.

Dr. Zanaty in a letter in your issue of Dec. 18th has explained what he means by "macroblast." Apparently he feels that since the distinction between "macroblast" and "megaloblast" is difficult in histological sections of bone-marrow, the "megaloblasts" in the bone-marrow of our cases of achrestic anæmia might have been "macroblasts." We agree that it is much easier to distinguish primitive cells of the normoblast series (macroblasts) from primitive megaloblasts in smears of sternal puncture material than in histological sections, but the histological picture of the femoral bone-marrow (which we examined in our cases) is highly characteristic in pernicious anæmia and, in our experience, quite easily distinguished from that of pseudo-aplastic anæmia; this difference is quite irrespective of terminologies and we refer Dr. Zanaty to the descriptions of Turnbull.[3] The histological structure of the bone-marrow in our cases of achrestic anæmia resembled that of pernicious anæmia and not that of the commoner pseudo-aplastic anæmia; hence our suggestions about its ætiology.

The occurrence of typical megaloblastic hyperplasia—as judged by sternal puncture material—in cases of megalocytic anæmia resistant to liver therapy has been reported by Dameshek and Valentine[4] (1937); we have noted such a case ourselves and correlated it with typical histological changes in the femoral marrow exactly like our reported cases of achrestic anæmia.

It will be seen that Dr. Zanaty's cases differ in many respects from our cases of achrestic anæmia; on the other hand, they fit very well the published descriptions of pseudo-aplastic anæmia, and we have little doubt that he has correctly diagnosed them as such. Since they are not examples of achrestic anæmia, we feel that his criticisms of our suggestions as to the ætiology of this anæmia are not valid.

We are, Sir, yours faithfully,

JOHN F. WILKINSON,
M. C. G. ISRAËLS.

Department of Clinical Investigations and Research, Royal Infirmary, Manchester, Dec. 20th, 1937.

MELANOSIS COLI

To the Editor of THE LANCET

SIR,—I was pleasantly surprised to receive a reprint from Sir Arthur Hurst of an article in *Guy's Hospital Reports* on melanosis coli[5] in which he states that "the chief causative factor is not constipation, nor conversion of a constituent of anthracene laxative into melanin, but the artificial diarrhœa produced by aperients of any kind taken on account of constipation." In 1933, in collaboration with J. H. Willard and Joseph Bank,[6] I reported 41 cases of melanosis coli diagnosed by sigmoidoscopy. Since Hurst quotes this article at length and minimises the significance of the anthracenes in their ætiological relationship to melanosis coli, I feel that some comment should be made on his argument. We found that in every patient from whom a detailed laxative history could be obtained (35 cases), one of the anthracene compounds had been used over long periods. It was stated at that time that the anthracene laxatives contain resinous substances and "in the form usually dispensed contain some pigment matter which seems intimately related to the active principle. It seems highly probable that the anthracene laxatives either contain pigment or elaborate a pigment within the colon, which is phagocytised by the deep mucosal cells, causing melanosis coli. Whether melanosis coli can develop in individuals who have not taken any of the anthracene laxatives it is impossible to state. The use of some of the laxatives in the anthracene group dates back to antiquity."

Hurst writes that "the diarrhœa results in excessive putrefaction of protein throughout the colon," and concurs in the opinion expressed by Pick that the melanin results from aromatic protein decomposition products of the contents of the colon under the influence of an oxidative ferment produced by the connective tissue cells of the mucosa. I do not see how it is possible to deny this mechanism for melanin deposits in the bowel, but he has added nothing to prove it. His claim that any aperient capable of causing diarrhœa may result in melanosis is based upon 2 case reports. The first patient had taken senna pods (an anthracene laxative) for sixteen years. No mention was made of the type of aperient used in the second case; apparently the essayist was unable to determine the laxative or laxatives employed. Obviously one cannot set aside a clinical experience in 35 patients with melanosis coli, who had used one of the anthracenes, on any such evidence. We analysed a separate group of 300 patients with constipation without evidence of melanosis. Only 12·3 per cent. of this group had used one of the anthracenes. Most of the patients had been taking non-anthracene laxatives for years, sufficient to cause a soft evacuation, yet melanosis did not occur.

Possibly the statistics of some observers giving a rather high incidence of melanosis coli, proximal to colonic carcinoma have been somewhat responsible for Hurst's views. Macroscopic evidence of melanosis coli in cancer of the colon has been exceedingly rare in our experience. Even though one admits its frequent occurrence in bowel cancer, there is nothing in the literature to indicate that the melanosis in these cases may not have resulted from the use of one of the popular anthracene laxatives.

Sir Arthur Hurst has not presented sufficient proof to cause one to abandon the part played by the anthracenes in the production of melanosis coli. It is my feeling that diarrhœa in association with ordinary putrefactive changes is probably not the cause of this condition.

I am, Sir, yours faithfully,

Philadelphia, Nov. 16th, 1937. H. L. BOCKUS.

WANTED SPECIMENS OF NORMAL SEMEN

To the Editor of THE LANCET

SIR,—The obtaining of material for research is a perennial difficulty. At the present moment I happen to be associated with Mr. B. P. Wiesner, Ph.D., on an investigation of changes in the semen occurring in various types of male infertility. Our chief difficulty at the outset is to obtain sufficient specimens of semen, to serve as "controls," from those who have produced families. I would like to bring particularly to the notice of the staffs of hospitals for

[3] Turnbull, H., in Vaughan's The Anæmias, 2nd ed. Oxford, 1936.
[4] Dameshek, W., and Valentine, E. H., *Arch. Path.* 1937, **23**, 159.
[5] Hurst, A. F., *Guy's Hosp. Rep.* 1937, **87**, 332.
[6] Bockus, H. L., Willard, J. H., and Bank, J., *J. Amer. med. Ass.* 1933, **101**, 1; *Lancet*, 1933, **2**, 877.

women that Dr. Wiesner will gladly undertake semen analysis of the husbands of hospital patients. Since a special technique is employed in the obtaining of specimens, it would be better that husbands should be sent to his laboratory, 37, Gt. Cumberland-place, W.1, so that they may be given instructions. It is hardly necessary for me to add that such work will be done gratuitously.

I am, Sir, yours faithfully,

KENNETH WALKER.

Harley-street, W., Dec. 22nd, 1937.

CHILD DISCIPLINE

To the Editor of THE LANCET

SIR,—Dr. Reginald Miller criticises my psychological methods in your Xmas issue. He takes up my method of curing a young thief by rewarding him for stealing: "Whether the plan works or not we should object to its principle, for it is merely an emotional reaction," says Dr. Miller. This to me is an astounding statement, for juvenile stealing is emotional; it is symbolic; the stolen money or trinkets symbolise the love that the child feels he hasn't got. By giving him a reward (that is a love token) I give the child what he is looking for and he doesn't then require to steal. The other way—the appeal to reason—does not work, naturally, for the fundamental motive is always unconscious. According to Dr. Miller it would appear that it was better to let a boy end in Borstal than to lower oneself to treat him in an emotional way. Yet in another paragraph I find Dr. Miller advocating treatment in an emotional way. On corporal punishment he writes: "It seems wrong to lay it down as a rule that no child should ever be whipped." He writes that after saying that corporal punishment "produces physical pain as well as emotional distress, and is apt to introduce a real element of fear," and he himself says that fear as a factor in routine training is to be avoided at all costs.

Again I find myself astounded. Would Dr. Miller punish a patient suffering from syphilis? Of course he wouldn't but he leads us to infer that he might punish a child who was suffering from a psychological disease. I suppose that the ideal of medicine is to find the root cause of disease and attack it. So, in dealing with a child, the teacher must try to find the root cause. Punishment never cures anything, because it never gets near root causes. Let me give a simple example. John, aged 15, lost his temper with the cook and swore at her hatefully. She complained to me. I sought John. "Got a letter from your mother this morning?" I asked. "Yes," he said, "how did you know?" "What did she write?" I asked. He snorted. "Choked me off cos I wrote her a letter in pencil instead of ink," he said in disgust. Here his anger took as object the best mother substitute—the food-giver. Suppose I had beaten him for his insolence to the cook. I could have made him fear, but his hate against interfering women would have remained unknown to him. My beating would have made him hate not only women but men. By making his action conscious I changed his attitude to the cook. I believe that this is the only possible way to deal with faults in children. Only, of course, the spanking or caning is much easier for the adult.

Dr. Miller is a humane man, yet I fear that his qualified approval of corporal punishment will simply encourage diehards to carry on with the stick. "Miller said it in *The Lancet*, and if he doesn't know, who should?" I am, Sir, yours faithfully,

A. S. NEILL.

Summerhill, Leiston, Suffolk, Dec. 28th, 1937.

CROYDON TYPHOID INQUIRY

TOWARDS the end of his opening statement reported in our last issue Sir Walter Monckton said "I have tried to assist you by marshalling the facts known to the corporation in an endeavour, whatever the consequences, to give you a full, candid and accurate account of the material upon which you have to work." And in conclusion he asked the tribunal to recollect that it is one thing to write field service regulations and lay down principles of perfection which the staff officer, the adjutant, and the commander in the field will endeavour to carry out and apply in practice, but it is quite another thing to determine whether the policy and whether the individual acts of those upon whom the responsibility of the real conflict rests is such that they have failed in the discharge of their duty to apply the principles laid down.

Before the officers of the corporation were called upon two expert witnesses were examined on the sources of the water-supply. Mr. W. S. Boulton, D.Sc., emeritus professor of geology at the University of Birmingham, gave evidence on the physical conditions under which water was obtained at Addington, and Mr. S. R. Rafferty, a consulting engineer, went into the question of the existing water resources.

DR. HOLDEN EXAMINED BY COUNSEL FOR THE CROYDON CORPORATION

Sir Walter Monckton began his examination of the medical officer of health, Dr. O. M. Holden, by taking him over the circumstances that led up to the outbreak. Among the early points which he cleared up was the fact that a case of paratyphoid was notified on Sept. 30th from South Croydon, all the later cases being typhoid except two, one of these quite recent and one in the middle of the outbreak. In reply to Sir Humphry Rolleston, Dr. Holden said that these were both paratyphoid B, whereas the recognised carrier was a typhoid carrier.

Dr. Holden agreed that he first discussed his investigations with the chief sanitary inspector on Oct. 30th and informed the chairman of the health committee that evening. On the same evening a Mr. Rimington called at his house and made suggestions as to dealing with possible causes of the infection which were noted by Dr. Holden; on the following day (Sunday) he went with the borough engineer to Mr. Rimington's house and later attended a meeting of local residents there. At that time six cases of typhoid had arisen in Croydon but he felt it might be inadvisable to make the fact public. "I have no authority myself," he added, "to issue statements." On Nov. 1st he telephoned one of the medical advisers at the Ministry and that morning he sent a sealed letter to some 26 doctors whose names were given in the local directory as situated in the South Croydon area. On the same day he asked the borough bacteriologist to let him have over the telephone the results of any blood tests (Widal) he had arrived at. On Nov. 3rd after a case of typhoid had been reported from Thornton Heath Dr. Holden notified all the doctors in Croydon whose names appeared in the local directory. On that same day there was also a meeting in the town clerk's room just after Dr. Conybeare's arrival from the Ministry. On Nov. 20th he received a message from the laboratory of the Ministry of Health that the excreta of one of the four men, who had been working intermittently in the well as a heading driver, had been found to contain the typhoid organism.

Sir Walter asked Dr. Holden how he was led to

advise chlorination of the Addington well water, which was done on Nov. 1st. Dr. Holden replied that he had discussed the subject with the borough engineer on Oct. 31st. "Up to that time I was under the impression that all the water-supply in Croydon was chlorinated. It surprised me to hear that that particular supply was not chlorinated, and I suggested that as it was the only unchlorinated supply it should be chlorinated." In giving this advice there were several factors that he was considering. There had been a long period of drought with the possibility that the draw in the well might have been extended on account of the pumping, and he had been looking up reports of the M.O.H. in 1905, '06, and '08, when tests had been carried out on the liability of the Addington well to surface pollution. "I knew of the conditions on the gathering ground and I had," he said, "for some time been a little uneasy about the Addington well."

Sir Walter then referred to some memoranda which had passed, the first being an exchange of letters in December, 1927, between the town clerk and Dr. H. P. Newsholme (who was then M.O.H.), in which Dr. Newsholme wrote:

"The analysis of water . . . cannot be regarded as satisfactory and it suggests some degree of recent contamination in the water collecting area; although the analysis is somewhat anomalous in view of the absence of bacillus coli. . . . I do not suggest here any special measures at the moment apart from continuance of the careful supervision of the filtration arrangements."

On Oct. 15th, 1930, Dr. Holden wrote to the borough engineer as follows:

"I notice in the analyses for drinking water that water from Addington shows bacillus coli in 10 c.cm. This is a higher number than is desirable, and it would appear as if some contamination either from animal or human sources is gaining access to this water. Should this persist it would be wise to consider the advisability of chlorinating the water before its distribution to consumers."

And on Sept. 18th, 1931, Dr. Holden wrote to the borough engineer:

"I notice . . . that the well at Addington shows the presence of bacillus coli in 10 c.cm. This well frequently appears to give this result and it would appear as if there is some soil contamination getting into the water. Should this area become built on to any greater extent and the introduction of cesspools multiplied this may lead to grave danger to the public health. May I suggest that in view of the tendency to the development of that part of Croydon the matter should be inquired into."

On May 6th, 1936, Dr. Holden wrote to the borough engineer remarking again on the presence of *B. coli* in 10 c.cm. of Addington water; "it would appear," he added, "that the filtration of this water is not entirely adequate." The engineer replied on the following day that the filters had been cleansed just before the taking of the sample and no doubt the result was due to the disturbance of the filter. ". . . as you are aware, we have a chlorination apparatus installed at Addington, and if you consider it necessary this could be put into operation, but I think, if possible, it would be better to avoid this."

On Nov. 7th, 1936, Dr. Holden wrote to the borough engineer:

"The results of the water analyses taken on Oct. 30th do not appear to be too satisfactory, more especially with respect to the Addington and Stroud-green wells. The best water appears to be that from Surrey-street. All the others, I feel, in view of the heavy rainfall we are having at the present time should be subjected to chlorination in order to avoid any possible accidents."

And the engineer replied on Nov. 9th: "With the exception of Stroud-green well, all the other supplies are being chlorinated."

These documents having been read to the tribunal, Dr. Holden agreed that he had in mind their contents in giving his advice which led to the Addington well water being chlorinated on Nov. 1st. Although he had advised chlorination at that time he was not then inclined to attribute responsibility to the Addington well, as he was then not quite sure of the area of distribution covered by the water from this well.

Continuing his examination of Dr. Holden on Thursday morning, Sir Walter Monckton returned to the decision reached on Nov. 4th by the public health committee not to make a public announcement then. The Addington well water had been chlorinated from Nov. 1st; on Nov. 4th the well ceased to pump except to Warlingham; on the following morning the water was chlorinated at the outlet of the reservoir. Dr. Holden said he regarded the chlorination undertaken as adequate if the infection was conveyed by water—"until midday on Nov. 3rd we were not at all sure that the infection was water-borne"—and thought that a public broadcast statement that all water should be boiled would cause unnecessary alarm. Circumstances had altered between Nov. 4th and 9th; there had been a large increase in the number of cases and on Nov. 9th what had been intended to be a statement in camera by the chairman of the public health committee was made public. On Nov. 4th he had notified all the doctors in the Croydon area.

Sir Walter then dealt with the extent and nature of the M.O.H.'s responsibilities in the borough. He is not summoned to meetings of the water committee and does not receive copies of its agenda or of its written minutes. His annual reports have not (nor have those of his predecessor) dealt with the water undertaking. He has not been called upon to prepare evidence for inquiries about the water-supplies. The monthly samples of water are selected by another officer of the corporation and submitted to the analyst. Dr. Holden said he had not been consulted about the work carried out by the water department and did not know what was being done on the Addington well until he learned of the fatal accident in the well through an account in the local press. Information had not been put before him about the plans of new houses in connexion with the gathering grounds.

Sir Walter read passages from the Sanitary Officers (Outside London) Regulations, dated Nov. 7th, 1935, which laid down (inter alia) that:

"A medical officer of health, in respect of the district for which he is appointed, shall (1) inform himself as far as practicable respecting all matters affecting or likely to affect the public health in the district and be prepared to advise the local authority on any such matter. . . ."

In response to Mr. Murphy, Sir Walter said he was not aware of anything in the regulations imposing on the medical officer of health any particular duties or responsibilities with regard to water-supply.

In reply to Sir Walter's question whether he had advised inoculation, Dr. Holden said he consulted with the bacteriologist and on his advice (and in confirmation of his own opinion) considered that as in all probability the infection had already taken place before they were aware of it, it would be risky to advise inoculation and would not in fact prevent the onset of typhoid. Dr. Holden added that at the meeting of the local profession on Nov. 10th the views put forward were diametrically opposed to one another.

On behalf of the Croydon division of the B.M.A., Mr. Forbes interposed to say that he made no point about inoculation. Had the medical officer conferred with the profession generally in the borough he would have found a division of opinion. Dr. Holden

remarked that one point put forward was that inoculation complicated the diagnosis of a case.

DR. HOLDEN EXAMINED BY HIS OWN COUNSEL

Mr. Sandlands (instructed by Messrs. Le Brasseur and Oakley for the London and Counties Medical Protection Society) questioned Dr. Holden about the increase in the duties of a medical officer of health during the last 20 years. Throughout the whole of that time water had never been put upon the M.O.H. except to advise when the reports were sent in. The year 1920, said Dr. Holden, seemed to be the last date on which the M.O.H. had anything to do with the water-supplies.

In reply to a question from Mr. Thesiger, Dr. Holden agreed that on Oct. 31st, when he learned from the borough engineer that the water at Addington well was not then being chlorinated, the borough engineer himself appeared to be very surprised at having found that this was so.

DR. HOLDEN CROSS-EXAMINED BY COUNSEL FOR THE SOUTH CROYDON TYPHOID OUTBREAK COMMITTEE

Mr. Lyons began by asking Dr. Holden about the limit of his present responsibility for the water-supply. "To advise the responsible authority on the bacteriological analyses," Dr. Holden replied, "as that is the only material put before me." He agreed he had made recommendations to the principal officer concerned—viz., the borough engineer. At the end of 1930 he had advised as regards one particular supply that the question of chlorination should be considered by the borough engineer. He was under the impression that chlorination at the Addington well had been in force since 1936. He had a memorandum from the borough engineer to that effect dated Nov. 9th, 1936, "in reply to your memo of the 7th inst. . . . with the exception of Stroud-green well, all the other supplies are being chlorinated." He was satisfied that what he had recommended was being done.

Mr. Lyons asked was or was not the water from Addington chlorinated before this trouble. Dr. Holden said he understood it was chlorinated intermittently. How long and for what purpose he did not know.

In reply to further questions Dr. Holden said he had examined from time to time the gathering grounds. He had never been asked by the water committee for advice on them. They had been the cause of some uneasiness of mind to him and he had watched continually the bacteriological analyses of water of this particular well.

Mr. Lyons then asked Dr. Holden if he accepted as a true statement of fact in his report for 1935 that "a constant supply of water was maintained throughout the year and has been satisfactory both in quality and quantity." That statement, said Dr. Holden, was given to him by the borough engineer. Mr. Lyons suggested that in view of the correspondence in 1931 Dr. Holden had allowed his report of 1936 to go out to the people of Croydon knowing that it was inaccurate and untrue. This Dr. Holden denied, and also the suggestion that he had omitted to say what he really knew.

(At this point the nature of Mr. Lyons's questions led Dr. Holden's counsel to point out that there was no absolute privilege in the tribunal and that anything said there might be the subject of an action later.)

Mr. Murphy now intervened to ask Dr. Holden whether a statement that "monthly analyses of the water were made at the five pumping stations and in many cases at more frequent intervals" was true in 1936, and Dr. Holden replied that the statement was not his but was given him by the engineer. It would not be accurate for 1937 but he had no reason to disbelieve that it was accurate for 1936.

Mr. Lyons then dealt with the question of cesspits, and asked Dr. Holden if it was correct to say there were only a few houses and bungalows in remote positions where the sewage went into such pits and Dr. Holden agreed. Hayling-grove was not remote but a cesspit there had not been used for four years. He knew of the cesspits in the Addington gathering grounds. It was perfectly correct under the Public Health Acts for a house to be erected with a properly constructed cesspit.

Questioned about the gathering ground Dr. Holden said he drew the attention of the responsible official to his ideas about that. He was not aware of the ground where four or five hundred people camped each week-end just outside Croydon borough. It was outside his area. He had heard of it now. There was no evidence that a nuisance was being caused by the encampment. He agreed that there was no convenience of any sort there but the distance was considerable between the camping ground and the well. Had he received information about the gathering ground, he would have watched the analyses in exactly the same manner as he now had done. Fisher's Farm was in such a position that he could not connect it up to any main drainage. These things had been there ever since (and he believed before) the well was sunk. His attention had been drawn to the pig farm about two and a half miles away at the beginning of the outbreak and he had been in collaboration with the medical officer of Purley about it.

Questioned about the unfortunate fatality to a workman in the well, Dr. Holden agreed that his first intimation was a press report of the accident. He was given to understand that when the fatality occurred work ceased. It would have been outside his province to say "This must stop, I must be in charge of this position." He had confidence in the borough engineer in charge of the work. If the engineer were in doubt he had not the slightest hesitation in thinking he would ask him.

Asked when he found out that work in the well had recommenced Dr. Holden said he did not find out directly; it was found out for him by Dr. Conybeare. When it came to his knowledge on Nov. 13th the work had already stopped. Men were working down the well from the end of September to the end of October. He did not know under what conditions they had been employed. "It is difficult for me," said Dr. Holden, "to know of work being carried on in a well unless I am told about it." "Do you not think," said counsel, "that the medical officer of health has power to give directions, not mere advice, on matters in which he thinks the public safety may reasonably be put in jeopardy?" "It depends," said Dr. Holden, "on the directions" and also, he added, on the internal working of the local authority.

Counsel: "I hope you recognise that in a matter of that nature there is no limit to the authority you have, or that any medical officer has."

Dr. Holden: "There are very definite limits. . . . I can only give advice to my committees."

Counsel: "I am afraid, if I may say so with every respect, you do not appreciate the position of a medical officer of health in all circumstances."

At this point Sir Walter Monckton interposed to ask whether Mr. Lyons was suggesting that by the operation of the Act of 1933 Dr. Holden was really

put in the position that he could give a direction over the heads of the committee.

Mr. Lyons: "In my submission, on a matter affecting the health of the people, the medical officer does not finish by saying 'I recommend so and so' and that is the end. I suggest that he says 'I direct in pursuance of the duty I have that this shall be done.' . . . I do not say that the medical officer can go out and pledge the corporation's credit for having the work done, but he can insist that the work shall be done, and he can bring the matter to the Minister of Health. He can go to the committee and say 'If you do not take notice I shall go to the Ministry.'"

Sir Walter Monckton: "That is not a direction."

Later on Mr. Lyons questioned Dr. Holden about the behaviour of a carrier. A carrier of typhoid, said Dr. Holden, may be in normal health and exhibit no observable symptoms. The Widal test was not absolutely conclusive but a very strong indication. A person might be a urinary carrier or a fæcal carrier and there were intermittent carriers.

Mr. Lyons questioned Dr. Holden about a conversation with Mr. Rimington and Mr. Moss on Oct. 31st when he was asked whether he thought the water-supply was a source of infection. He certainly did not use the word "inconceivable" but might have said that he could not suspect water until he had eliminated other factors. At that time the borough engineer was under the impression, as he was, that the water was chlorinated.

In answer to a later question Dr. Holden said he would like to emphasise that in any outbreak of typhoid or paratyphoid there were many factors to take into consideration. Water, food, personal contact, or personal relationships all had to be considered—and on Oct. 31st when four cases had arisen there was no evidence which could direct him in particular to incriminate any article either of food or drink.

Further cross-examined on Dec. 28th, Dr. Holden stated that the borough engineer, like himself, was under the impression that chlorination had been continuous. He could not himself find the exact dates of chlorination. He gave no instruction to sample tap water in the houses from which four cases were notified on Oct. 30th; water was not always the most obvious cause of typhoid. He did not warn the matron of a nursing-home in St. Augustine's-avenue which obtained its water from the high level source; each patient there had a private doctor and the doctors had been warned. He did not agree that the measures taken against the danger were unsuccessful; even 300 cases were a comparatively small figure for a water-borne outbreak; he had expected more. Replying to Mr. Forbes, who represented the local division of the B.M.A., Dr. Holden said that his letter to local doctors on Nov. 4th said nothing to indicate that the source of infection was water, because he was not sure.

HEALTH OF THE NAVY

AN improvement in the Navy's general health is shown by a decrease in the returns for disease and injury in the annual report for 1935,[1] and the number of men sick daily was the lowest on record—namely, 1569 (18·17 per 1000) in an average total strength of 86,345. The lowest sick-rate was in the America and West Indies station and the highest in the Irregular list, which includes the crews of warships on passage to or from foreign stations, thus containing a large proportion of invalids. Altogether 1304 men were invalided, an increase on the previous year and on the preceding five-year period; of these, 13·94 per 1000 were due to disease and 1·15 to injury. This total also includes 937 men finally invalided (10·85 per 1000); pulmonary tuberculosis was the commonest cause, accounting for 169; errors of refraction came second with 112, and injuries third with 100.

The specialist medical officers are devoting particular attention to diseases of the stomach and intestines, which has been steadily increasing for some years. There were 2944 cases in all, gastritis, gastro-enteritis, and enteritis being responsible for 1605, and appendicitis for 449. The common cold, influenza, and tonsillitis are still the commonest causes of sickness and cost the Navy some 160 years' loss of service in the twelve months. The standards for eyesight are being relaxed further and now spectacles may be worn on duty by all except officers of the executive branch and ratings of the seaman and signal branches. With these concessions and the more precise methods of examination it is expected that wastage due to sub-standard eyesight will be even more reduced. Though the incidence of pulmonary tuberculosis is very much lower than in the groups examined in the merchant navy, the position is still unsatisfactory. The problem is complex and is being investigated in conjunction with the Medical Research Council and the Royal College of Physicians of London. Unremitting attention is being paid to the selection of personnel, to the environment on board ship, and to the diet. Modern methods of diagnosis may help to eliminate candidates showing the early signs and to detect the disease among the serving personnel, but the effects are by no means certain.

The incidence of venereal disease continues to fall, with 254 fewer fresh cases of gonorrhœa than in 1934, the total being 3572, but new cases of syphilis remained stationary. The China station was the worst affected, the proportions per 1000 being 100·11 for gonorrhœa and 93·56 for syphilis. Fungoid infections of the feet (epidermophytosis, "athlete's foot") are increasing and becoming geographically more widespread; all but the mildest cases require prolonged and careful treatment. In some ships abroad as many as 45 per cent. of the complement were infected. Preventive measures include suitable footwear in hot stations, steam laundering of towels, tiling of bathrooms, frequent inspection to detect early cases, and lectures to the men on foot hygiene. In the Persian Gulf, through encouragement to the men to work barefoot the incidence declined and there was also a readier response to treatment. The measures recommended for disinfecting fresh fruit and vegetables—immersion in boiling water for 30 sec. or in strong potassium permanganate for 30 min.—have been found to be ineffective. They are considered likely to produce an ill-founded sense of security and it is urged that all fresh vegetables should be cooked.

There were 6125 surgical operations in the naval hospitals or in the hospital ship *Maine*, besides a considerable number in ships at sea and in other shore establishments. The increasing use of the Winnett Orr method of immobilising compound fractures and osteomyelitis produced excellent results and economy of staff, time, and dressings. Evipan has proved a valuable anæsthetic for medical officers afloat, especially in single-handed operations.

The total death-rates for the last two years were the lowest on record, being 1·96 per 1000 in 1935. Pneumonia (13) and pulmonary tuberculosis (15) and appendicitis (10) head the list of diseases, but violence was responsible for 48 (25 from wounds, fractures, &c., 13 from suicide, and 8 from drowning).

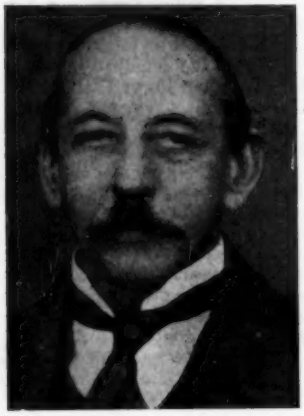
[1] Report on Health of the Navy. H.M. Stationery Office. 2*s*. 6*d*.

OBITUARY

GEORGE ALFRED BUCKMASTER, D.M. Oxon., F.R.C.S. Eng.

Prof. G. A. Buckmaster, who has been described as one of the last of the great Edwardians in scientific medicine, actually spent his early professional life in Victorian days; for he was 78 when he died last week, though he looked much younger. The son of John Charles Buckmaster of the Science and Art Department, South Kensington, George Alfred was as a boy the pickle of a large family whose members were destined to acquire fame: one of his brothers became Lord Chancellor of England, one a distinguished educationist, another a well-known painter.

George Buckmaster went to Magdalen College School, where his eldest brother was assistant master, and thence, in 1877, as a demy to Magdalen College, Oxford. He did well in natural science, obtaining a first-class in 1881, and was awarded the Burdett-Coutts scholarship in geology in 1882. He studied medicine at St. George's Hospital, qualifying in 1883, and for the next three years he held a Radcliffe travelling fellowship, working at various foreign universities, including Leipzig, Kiel, Göttingen, and Berlin. One of his earliest pieces of research work, on tetanus, was published in a German journal. He took his Oxford medical degree in 1885, and became D.M. in 1887, acquiring a D.P.H. in the same year. His scientific equipment fitted him for selection as one of the three English members of the Leprosy Commission in India, 1890–91. This commission reported in 1892 and its sane conclusions and suggestions might be studied with advantage even to-day.



PROF. BUCKMASTER

On his return home George Buckmaster started his career in physiology, becoming a professor on the staff of the Royal Veterinary College and an assistant professor at University College, London, where he soon achieved popularity as a vivid and stimulating lecturer. Students appreciated his inexhaustible fund of anecdotes about the pioneers of physiology and the wide range of his general as well as his special reading. His main interest at that time was in the morphology of blood—he held strongly to the belief that blood platelets were artefacts—and in its function, especially the mode of carriage of oxygen and carbon dioxide. His monograph on the "Morphology of the Blood" (1906) was acclaimed as a distinguished contribution to the subject. He collaborated with J. A. Gardner in work on blood gases and on the physiology of anæsthesia, mostly published in the *Proceedings* of the Royal Society. With Gardner he invented a neat device in the shape of a blood gas pump which had no taps and therefore could not leak; by means of this instrument they were able to show that the amount of free nitrogen in blood is small but constant.

In 1919 Buckmaster succeeded Stanley Kent in the chair of physiology at the University of Bristol, where he soon won the affection and admiration alike of students and colleagues. He kept this appointment until he retired from active professional life in 1930, retaining only his examinerships. In 1934 he was made F.R.C.S. Eng. and was immensely proud of this honour, which he had earned as a devoted and faithful servant of the college; he twice made the journey to Australia and New Zealand as an examiner when he was already over 70, and only two years ago he visited India to conduct the physiology part of the primary fellowship examination in Calcutta. A co-examiner on such visits expresses the admiration everywhere felt for this "marvellous old man" whose contacts had been so many and so wide, and the conviction that no guest had ever been more welcome in either the social clubs or the academic circles of Australia and New Zealand. Buckmaster's prodigious memory, his brilliant talk, and the clever stories he told would indeed hold spellbound an audience of any profession or rank. He had an astonishing fund of information and very varied interests; for instance a visitor to his laboratory at Bristol one day found him trying to rear a family of glow-worms on a plentiful diet of snails. He was fond of an argument up to a point; if it seemed to go against him he would jump up and fetch the violin on which he played with almost professional skill. As a letter-writer he was superb, and in this as in other qualities he seemed to belong to a fuller and more leisured age.

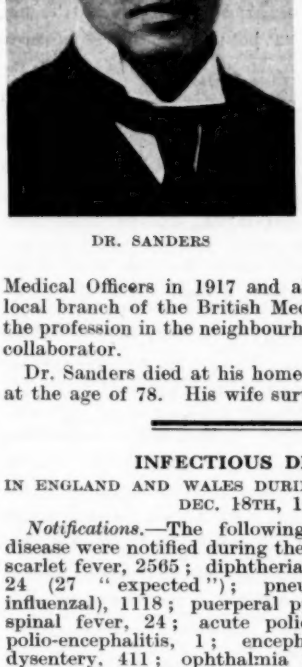
A friend writes: "Buckmaster abhorred intellectual dishonesty or even pretentiousness, and he had nothing but scorn for spurious claims to knowledge. Around him gathered a few cronies, distinguished like himself in scholarship and erudition. One of the closest of these, Prof. William Wright, died only a few months before him. He relished also the company and friendship of a younger set to whom he stood as a wise and benevolent mentor, dignified with the atmosphere of other times and other places, It was a tragic irony that with him, as with Baudelaire, an aphasia should have marred the closing days of one who so loved speech and possessed such mastery over language."

George Buckmaster married Amy Elizabeth, daughter of Charles Brooks, who survives him with one married daughter. His only son died in Ireland just after the war.

CHARLES SANDERS, M.B. Lond.

DURING long tenure of office as medical officer of health for the London borough of West Ham Dr. Sanders worked devotedly for his locality. Qualifying from St. Bartholomew's Hospital in 1881, he held resident posts at the Queen's Hospital, Birmingham, and was appointed to West Ham in 1887. The 37 years in which he controlled his department were years of rapid expansion, in which the population rose from 186,000 to 314,000, and in which the administrative side of public health increased out of all knowledge in complexity. The leading characteristics of Sanders as a chief during this formative period of the health services were his cheerfulness, his capability, and his fair dealing. One of his staff writes of his energy and his readiness to sacrifice

his own pleasure and leisure, and of the confidence his strong personality inspired; the entire staff, he says, felt at all times that they could be sure of his treating them justly. He was always ready with sound advice for those who asked it, and he won the affection of those that worked with him. On his retirement in 1924 he received warm testimonials from the public health department and the school medical service. Besides acting as M.O.H. and S.M.O. he formerly held the appointments of surgeon to the West Ham Hospital and medical officer to the London and India Docks Joint Committee. He was elected president of the Society of Medical Officers in 1917 and also presided over the local branch of the British Medical Association. To the profession in the neighbourhood he was a welcome collaborator.



DR. SANDERS

Dr. Sanders died at his home at Kew on Dec. 18th at the age of 78. His wife survives him.

INFECTIOUS DISEASE

IN ENGLAND AND WALES DURING THE WEEK ENDED DEC. 18TH, 1937

Notifications.—The following cases of infectious disease were notified during the week: Small-pox, 0; scarlet fever, 2565; diphtheria, 1568; enteric fever, 24 (27 "expected"); pneumonia (primary or influenzal), 1118; puerperal pyrexia, 153; cerebro-spinal fever, 24; acute poliomyelitis, 10; acute polio-encephalitis, 1; encephalitis lethargica, 2; dysentery, 411; ophthalmia neonatorum, 81. No case of cholera, plague, or typhus fever was notified during the week.

The number of cases of dysentery notified during this week is 30 less than last and 44 less than two weeks ago. A large proportion of the cases were reported from the home counties—e.g., London 127, Middlesex 43, Essex 33, Sussex West 14, Surrey 10, Kent 8, Sussex East 7; other counties with considerable numbers being Devon 42, Notts 22, Norfolk 21, Staffs 18, Chester 14, Lancaster 12, Glamorgan 11.

The number of cases in the Infectious Hospitals of the London County Council on Dec. 24th was 4077, which included: Scarlet fever, 801; diphtheria, 1512 (diphtheria carriers, 30); measles, 392; whooping-cough, 349; puerperal fever, 12 mothers (plus 8 babies); encephalitis lethargica, 283; poliomyelitis, 3. At St. Margaret's Hospital there were 19 babies (plus 6 mothers) with ophthalmia neonatorum.

Deaths.—In 125 great towns, including London, there was no death from small-pox, 6 (5) from enteric fever, 22 (0) from measles, 4 (0) from scarlet fever, 22 (8) from whooping-cough, 49 (6) from diphtheria, 57 (15) from diarrhœa and enteritis under two years, and 52 (11) from influenza. The figures in parentheses are those for London itself.

Five deaths from typhoid were reported from Croydon, 1 from Portsmouth. Liverpool had 7 fatal cases of measles, Manchester 4. Liverpool also reported 7 deaths from whooping-cough. Diphtheria was fatal in 29 great towns, Liverpool reported 8 deaths, Hull and Sheffield each 3.

The number of stillbirths notified during the week was 294 (corresponding to a rate of 46 per 1000 total births), including 38 in London.

MEDICAL NEWS

University of Cambridge

At recent examinations the following candidates were successful:—

THIRD EXAMINATION FOR M.B., B.CHIR.

Part I, Surgery, Midwifery, and Gynæcology.—J. Arnott, J. L. W. Ball, C. H. Bartlett, A. H. Baynes, J. R. Bignall, G. Bourne, R. I. C. Bradford, B. R. Bray, R. W. W. Brown, L. H. Cane, D. M. Carding, I. C. Chopra, A. C. Clark, M. J. Clow, J. Clutton-Brock, P. C. Conran, E. Cronin, A. J. Daly, E. B. Davies, C. H. C. Dent, A. P. Dick, W. D. Doey, K. W. Donald, G. W. N. Dunn, N. B. Eastwood, T. A. W. Edwards, G. P. Fox, E. K. Gardner, O. D. Gilmore, M. H. Harmer, W. J. Hay, J. S. Heller, H. G. W. Hoare, R. D. Holloway, G. N. Hunt, H. M. James, R. M. Johnstone, E. P. Jowett, P. A. Knill Jones, J. Laughlin, J. W. Lillico, G. M. Little, A. G. Marshall, B. E. Miles, R. O. Murray, J. C. B. Nesfield, G. F. Panton, J. S. Phillpotts, J. G. B. Platts, B. H. Price, J. S. Prichard, G. C. Pritchard, J. H. B. Round, D. Russell-Davis, J. Smith, P. M. Smythe, H. Stott, J. Sutcliffe, R. J. D. Temple, J. R. T. Turner, A. J. Walton, J. M. Watson, H. F. Whalley, D. O. Wharton, B. D. Whitworth, E. H. L. Wigram, M. M. Wilson, J. W. Wood, P. J. Wormald, G. M. Wright, and J. Yudkin.

Part II, Physic, Pathology, and Pharmacology.—G. E. Adkins, D. A. P. Anderson, T. L. Barbour, C. F. Barwell, R. D. I. Beggs, A. S. Bookless, A. W. Box, J. B. Bunting, G. A. Burfield, A. L. Cochrane, S. B. Darbishire, J. K. Denham, W. D. Doey, E. W. Dorrell, L. C. de R. Epps, T. J. Fairbank, A. L. Fawdry, W. Goulstone, J. L. Griffith, E. B. Hacking, S. W. G. Hargrove, F. E. S. Hatfield, H. G. W. Hoare, J. H. S. Hopkins, C. W. Hutt, R. T. Johnson, H. S. Kellett, G. J. G. King, A. P. Kitchin, J. I. H. Laurie, S. A. H. Lesser, R. W. Mason, R. M. Miller, K. S. Mullard, T. L. Oliver, B. G. Parsons-Smith, G. R. C. Peatfield, P. A. T. Phibbs, A. L. Phillips, H. L. Porter, C. G. Rob, D. Rubin, G. Sheers, J. Sutcliffe, V. E. A. Sykes, R. D. Teare, D. J. D. Torrens, R. L. Townsend, R. C. H. Tripp, S. C. Truelove, N. Vere-Hodge, J. H. Ward, S. R. F. Whittaker, J. M. G. Wilson, M. C. Woodhouse, G. H. Wooler, and J. D. Younghusband.

University of London

At recent examinations the following candidates were successful:—

ACADEMIC POST-GRADUATE DIPLOMA IN CLINICAL PATHOLOGY

Tatjana von Haebler.

EXTERNAL DIPLOMA IN CLINICAL PATHOLOGY.

C. E. W. Hoar.

THIRD EXAMINATION FOR THE B.D.S.

R. L. Kenyon and A. D. Renson.

Society of Apothecaries of London

At recent examinations the following candidates were successful:—

Surgery.—F. B. Akeroyd, Univs. of Camb. and Leeds; J. S. Lancaster, Univ. of Camb. and St. George's Hosp.; and J. D. B. Perkins, Guy's Hosp.

Medicine.—F. Bastawros, Royal Colleges, Edin.; F. C. E. Diamond, Univ. of Durh.; and D. C. Light, Guy's Hosp.

Forensic Medicine.—F. Bastawros, Royal Colleges, Edin.; F. C. E. Diamond, Univ. of Durh.; and D. C. Light, Guy's Hosp.

Midwifery.—J. M. Hardy, Univ. of Manch.; P. H. Hay-Heddle, Westminster Hosp.; and R. Lipkin, Univ. of Liverp.

The following candidates, having completed the final examination, are granted the diploma of the society entitling them to practise medicine, surgery, and midwifery: F. Bastawros, F. C. E. Diamond, and D. C. Light.

National University of Ireland

At recent examinations at University College, Cork, the following candidates were successful:—

M.D.

Thomas O'Neill.

FINAL EXAMINATION FOR M.B., B.CH., B.A.O.

J. C. Dundon (first class honours); J. A. Allen, Patrick Buckley, Patrick Coffey, J. C. Dundon, K. A. Flaherty, F. S. Kelleher, D. J. G. Maher-Loughnan, Coleridge Marchment, E. F. Mulcahy, Ellen M. M. Murphy, Patrick Murphy, J. S. O'Mahony, and P. J. Ryan.

Part I.—Mary Corbett, John Hutch, Loretto Kelly, D. J. R. McConvell, David O'Keeffe, Patrick O'Keeffe, and Mary F. O'Leary.

Part II.—C. J. Clohessy, G. R. Cubitt, James Fitzgerald, J. G. Paton, and M. F. Ronayne.

Royal Society of Medicine of Budapest

Mr. Leslie Paton has been elected an honorary member of this society on the occasion of the centenary of its foundation.

University of Bristol

At recent examinations the following candidates were successful :—

FINAL EXAMINATION FOR M.B., CH.B.

Section II.—R. L. J. Derham, H. D. T. Gawn, M. E. M. Herford, F. J. W. Hooper, G. L. Page (with distinction in public health), and Anna H. Silberstein.

Group II, completing examination.—P. N. Heron and P. K. Jenkins.

L.D.S.

H. B. Harding and J. C. F. Rolls.

Queen's University, Belfast

Dr. Richard Hunter has been appointed secretary of the university.

Demonstrations of Contraceptive Technique

On Thursday, Jan. 6th, at 2.30 P.M., a practical demonstration of the technique of the use of a variety of contraceptive methods will be given at the C.B.C. Clinic by Mrs. Marie Stopes, D.Sc., and Dr. Evelyn Fisher. Medical practitioners and senior students should apply for tickets to the hon. secretary, C.B.C., 108, Whitfield-street, London, W.1.

Regional Coördination of Voluntary Hospitals

The Voluntary Hospitals Commission recommended the division of the country into hospital regions, the formation of regional councils to correlate their work and needs, and the establishment of a central council to coördinate the work of the regional ones. The British Hospitals Association has set up a provisional central council to advise how these recommendations may best be carried out. The members will include Mr. S. P. Richardson (chairman), Mr. Arthur Morley, K.C. (vice-chairman), Mr. R. G. Hogarth, F.R.C.S., Prof. A. W. Sheen, F.R.C.S., and two representatives to be appointed by the British Medical Association. Mr. R. H. P. Orde is the hon. secretary.

The Oxford and Bath Congresses on Rheumatism and Hydrology

The International Congress on Rheumatism and Hydrology, which is to be held in Oxford from March 25th to 31st, has been convened by the International Society of Medical Hydrology and the International League against Rheumatism. The president-elect is Sir Farquhar Buzzard and the chairman of the British organising committee Sir Humphry Rolleston. There will be two main subjects for discussion. The first, wet and dry climates and weather in the causation of disease, will be introduced by Prof. G. Danischewsky (U.S.S.R.), Dr. G. Edström (Sweden), Prof. de Rudder (Germany), and Dr. E. P. Poulton. The second, juvenile rheumatism, will be considered under four aspects, and the principal speakers will include Prof. F. Klinge (Germany) and Dr. A. G. Gibson, pathological aspect; Prof. L. Findlay and Dr. H. Grenet (France), clinical aspect; Dr. Wilfrid Sheldon, therapeutic aspect; and Dr. F. J. Poynton and Prof. Danischewsky, social aspect. Visits will be paid to hospitals in London on March 26th. Arrangements are being made for the accommodation of congress members in colleges at Oxford, and there will be visits to Leamington Spa during the meeting and to Droitwich at the end. The congress is open to medical men and scientists who are not members of either body. Inquiries should be addressed to the general secretary at 109, Kingsway, London, W.C.2.

The Bath Bicentenary Congress on Chronic Rheumatism is to be held to commemorate the bicentenary of the Bath Royal National Hospital for Rheumatic Diseases from March 31st to April 3rd. It will thus follow directly after the international meeting at Oxford, and the speakers are to include authorities on rheumatism from France, Germany, Holland, Sweden, and other countries. The meeting will be opened by Lord Horder on the Thursday evening, and the following day will be devoted to a discussion on gout and rheumatism of metabolic origin. On Saturday papers will be read and next morning there will be a demonstration of cases at the hospital. The meeting will be open to all practitioners, and further particulars may be had from the honorary secretary of the congress at the Royal National Hospital for Rheumatic Diseases, Bath.

Broadcasting Talks

On Jan. 7th the first of nine Friday morning talks on sickness in the house will be given. The talks will be given by doctors and nurses, and will deal with the care of invalids and old people. Later there will be three talks on the problems of adolescence.

Appointments

BELL, R. B., M.R.C.S. Eng., Assistant (Deputy) Medical Officer at New Cross Hospital, Wolverhampton.

DAVIS, W. B., M.D. Belf., D.P.H., Pathologist to the Victoria Hospital, Blackpool.

DIXON, IRENE, M.B. Lond., D.P.M., Assistant Woman Medical Officer in the Headquarters Branch of the Post Office.

GILL, T. P., M.B. Aberd., F.R.C.S. Edin., D.L.O., Hon. Surgeon to the Ear, Nose, and Throat Department of the Kent County Ophthalmic and Aural Hospital, Maidstone.

HARDING, H. E., F.R.C.S. Eng., Clinical Assistant to the Fracture and Orthopædic Departments of King George Hospital, Ilford.

HILL, T. R., M.D., M.R.C.P. Lond., Diagnostic Physician to the Tavistock Clinic, London.

LEWIS, R. I., M.D. Lond., Medical Superintendent at the Hospital for Tropical Diseases, London.

MCCARTAN, WILLIAM, M.D. Belf., M.R.C.P. Lond., D.P.H., D.P.M., Medical Superintendent at the Brighton County Borough Mental Hospital, Haywards Heath, Sussex.

PRIEST, P. D., L.D.S. R.C.S., Dental Registrar at the London Hospital.

RAE, L. J., M.B. Camb., D.M.R.E., Assistant Radiologist in the London Hospital.

SANDIFER, P. H., M.R.C.P. Lond., House Physician at the National Hospital, Queen-square, London.

SMITH, E. R., M.D. Liverp., D.P.H., District Tuberculosis Officer for Cheshire.

STEWART-WALLACE, A. M., M.D., M.R.C.P. Lond., Resident Medical Officer at the National Hospital, Queen-square, London.

UTTING, ERCENWIN A., M.R.C.S. Eng., Assistant Woman Medical Officer in the Headquarters Branch of the Post Office.

VICK, R. M., M.Chir. Camb., F.R.C.S. Eng., Hon. Consulting Surgeon to the Bexhill Hospital.

London County Council Hospital Staff.—The following appointments are announced. D.M.S. (I) or (II) = Deputy Medical Superintendent Grades I or II. A.M.O. (I) = Assistant Medical Officer Grade I.

DORLING, G. C., F.R.C.S. Eng., D.M.S. (I), Lewisham;

PRICE, I. I., M.B. Lond., F.R.C.S. Eng., D.M.S. (I), St. Mary, Islington;

GABE, JOEL, M.B. Lond., F.R.C.S. Eng., D.M.S. (II), St. Andrew's;

WIMBUSH, H. G., B.A. Camb., M.R.C.S. Eng., D.M.S. (II), Hammersmith; and

JONES, A. N., M.B. Liverp., F.R.C.S. Eng., D.C.O.G., Senior A.M.O. (I), St. Pancras.

Queen Charlotte's Maternity Hospital, London.—The following appointments are announced :—

DICKINSON, J. R., M.B. Camb., Assistant Resident Medical Officer;

GLENNIE, EVELYN W., M.B. Glasg., Resident Anæsthetist and District Resident Medical Officer; and

REED, GRACE E., M.B. Lond., Resident Anæsthetist.

Births, Marriages, and Deaths

BIRTHS

PATERSON.—On Dec. 27th, the wife of Dr. A. S. Paterson, Cheyne Walk, Chelsea—a son.

ROCHE.—On Dec. 25th, 1937, at 7, Knaresborough-place, S.W.5, the wife of Alex. E. Roche, F.R.C.S.—a daughter.

ROWE.—On Dec. 21st, at Blackheath, to Dr. C. Isobel Rowe (née Pattullo), wife of Flight Lt. F. C. T. Rowe—a son.

MARRIAGES

DIXON—TABOR.—On Dec. 21st, at S. Mary, Bocking, John Hardy Dixon, M.B., to Winifred Hope, younger daughter of E. H. Tabor, of Bocking, Essex.

FAIRWEATHER—DUFFUS.—On Dec. 18th, at St. Nicholas Parish Church, Harpenden, Josiah William Chalmers Fairweather, M.B., to Helen Vera Mary Duffus, widow of James Logie Lyall Duffus.

DEATHS

BRAMHALL.—On Dec. 25th, in London, after a long illness, Lt.-Col. Charles Bramhall, R.A.M.C., retired, O.B.E., M.R.C.S. Eng., of Brook Meadow, Warsash, Hants.

BUTLER.—On Dec. 25th, at Belmont, Guildford, Harold Branson Butler, M.B.E., F.R.C.S., Edin., Consulting Surgeon to the Royal Surrey County Hospital, Guildford.

DAY.—On Dec. 20th, at Harleston, Norfolk, Donald Douglas Day, M.B. Lond., F.R.C.S. Eng., aged 79.

MCDOWELL.—On Dec. 23rd, at Mowbray, Cape Town, Laurence Fitzhenry McDowell, M.D. Dubl.

STARKEY-SMITH.—On Dec. 23rd, at Hungerford, Thomas Gordon Starkey-Smith, M.D. Lond., aged 57.

WARDROP.—On Dec. 25th, at Torbane, Camberley, Douglas Wardrop, C.B., C.V.O., M.B. Aberd., retired Colonel A.M.S.

N.B.—A fee of 7s. 6d. is charged for the insertion of Notices of Births, Marriages, and Deaths.

NOTES, COMMENTS, AND ABSTRACTS

THE SOCIAL CARE OF THE EPILEPTIC *

By RALPH H. CROWLEY, M.D., F.R.C.P. Lond.
LATE SENIOR MEDICAL OFFICER TO THE BOARD OF EDUCATION

HE would be a bold man who claimed he had ever cured a case of so-called idiopathic epilepsy in its well-established form. He may claim to have controlled the number and severity of the fits with commensurate relief to the patient. Nevertheless I know by long experience how depressing can be the critical survey of the results of treatment of children in residential schools for epileptic children. And there is evidence that were we to follow up the adolescents when they leave school the results would depress us still more. Dr. Tylor Fox's experience at Lingfield Colony has made clear the waste that ensues from the failure to follow up effectually the children leaving the colony school to take up life at home. Equally unsatisfactory is the lot of the large proportion of children for whom provision is not available at any such colony. But while it was likely that the disabilities from which both these classes of children suffered would increase rather than diminish as the child passed into later adolescent and adult life, little accurate knowledge has been available about the life of the epileptic in the community and the difficulties encountered by him and his family at home. There seemed then to be a definite need for a small-scale inquiry into this social aspect of epilepsy and for the working out of a practical scheme of help for the epileptic. The Central Association for Mental Welfare, with whose work I am closely associated, agreed to carry out such an inquiry.

A SAMPLE CENSUS

Accordingly in the summer of 1936, under the expert guidance of Dr. Tylor Fox, a preliminary inquiry was instituted in Essex in the districts of Barking, Romford, Leyton, Walthamstow, and Dagenham. The population of this area was 475,979. The names of 565 cases were received from the local authorities, hospitals and institutions, and from private individuals. From the records available the mental and social status of each patient was determined as far as practicable, and 57 patients were selected for home visits. The incidence of epilepsy in the area as shown by this inquiry was 1·2 per thousand, but it is probable that if the figures from the London Hospital could have been included some 250 cases would have been added and the incidence raised to about 1·8 per thousand. A rough classification of the cases was made into "sane," "unstable," and "mentally defective." The term "sane," though open to objection, was retained as it has an accepted meaning when applied to epileptics. The figures for epileptics of normal intelligence are probably too high, as the high-grade feeble-minded epileptic tends to be undetected in a busy out-patient clinic. The "unstable" group included those with well-marked manifestations of temperament peculiar to epileptics, and those who had been admitted to mental hospitals. No information was obtained in 155 cases of the 565. Of the remaining 410, 234 were classified as "sane," 45 as "unstable," and 131 as "mentally defective."

The inquiry was later extended to Tottenham and Wood Green and, as circumstances were particularly favourable, to Ipswich. In Ipswich the help of a large proportion of the practitioners in the borough was obtained through the local secretary of the British Medical Association, and this brought to light a much larger number of epileptics than in the previous areas. The total number reported was 225 out of a population of about 92,000, giving an incidence of 2·44 per thousand. Of the 225 cases, 162 were non-institutional and 63 institutional; 32 were 50 years of age or over; 145 were between 16 and 50 years; and 48 were under 16 years. Among the 162 non-institutional cases, 99 were reported sane, 30 unstable or deteriorating, and 19 mentally defective. The mental condition of 14 patients was unrecorded. Among the 63 institutional cases the corresponding numbers were 7 sane, 24 unstable or deteriorating, and 22 mentally defective. In addition 10 were reported as insane.

HOME VISITS

From the reports received on the visits to patients' homes it was evident that the young epileptic was often a great burden upon the mother, and parents were thankful to discuss their problems with someone who could advise them. Many were found to be worrying about the future of their children. The mothers tended either to keep their children in cotton-wool or to allow them to run serious risks. The post of errand boy on a bicycle was considered a desirable one by some mothers, and one allowed her son to become a painter with the result that he fell 20 ft. through a window. Another mother, on the other hand, would not let her son leave the house alone although he had only two or three fits a month. It was evident that friendly advice and assistance in placing children in suitable occupations would be very welcome. An opportunity for mothers to recount their worries, some suggestions for surmounting their difficulties, and the feeling that disinterested advice is available in a crisis, all help to mitigate the burden of solitary and unending responsibility. Normally a mother has little chance of a respite, and both she and her child suffer from being debarred from convalescent and holiday homes.

Careful inquiry was made on the question of employment. It was found that in Essex and to a less extent in Ipswich, the majority of adult epileptics were unemployed and bitterly resentful of the fact. The most pitiable cases were those men, apparently of normal intelligence and with no obvious signs of degeneration, who were unable to get work either because their doctors had forbidden it, although they were told to occupy themselves, or because their epilepsy was known and employers were frightened of it. The unemployed were mainly men, as female epileptics, even when unmarried, have household duties, and there are more safe and sedentary occupations open to them. But life is seldom easy for either sex, and most of the adult patients seemed to be suffering from a sense of isolation, hopelessness, and depression almost to the point of despair. Some remarked that they had fewer fits when they had work to occupy their minds. Moreover forced inaction makes them more difficult, and the corresponding increase in fits produces a greater strain on all concerned. It is indeed a vicious circle.

A PROBLEM IN MENTAL HEALTH

The inquiry showed most clearly that treatment of the epileptic, apart from the control of fits, is a

* Abstract of a paper read at the annual meeting of the International League against Epilepsy (British Branch) on Oct. 22nd, 1937.

matter of attending to the mental health both of the patient and of his family. A full recognition of this would profoundly modify our present methods. The doctor, whether as private practitioner or hospital out-patient physician, has but a limited knowledge of the social and industrial circumstances of his patients, and few opportunities of exploring their symptoms of mental ill health and maladjustments. The epileptic needs all the advantages that the psychiatric clinic is bringing to the psychoneurotic, together with the assistance in the home that the mental welfare worker can render. The hospital doctor requires the help of the mental welfare worker as surely as the medical officer of health and the school medical officer require that of the health visitor and the school nurse. The hospital doctor, the private practitioner, the mental welfare worker, and the after-care visitor acting as a team and backed by the community could bring untold relief to the daily lot of the epileptic. Such a welfare organisation must operate early if it is to prevent the maladjustments of adult epileptics, for some 80 per cent. of cases have their origin in childhood and adolescence, and mental disabilities which begin then often steadily increase through adult life. On these lines we might hope to do for the epileptic very much what has been done for the cripple and is being done for the child with rheumatism and early heart disease, and prevent much of the "crippling" due to epilepsy.

FOLLOWING UP

The Central Association for Mental Welfare intends to follow up the work thus begun. It may be advisable to extend the inquiry to other types of areas before formulating a general line of action, but in the meantime much will be learned by observing the progress of the patients and families already visited. As the immediate result of the inquiries so far undertaken the Association is considering:—

(*a*) The extension to young epileptics leaving residential schools and colonies of their present system of after-care, and the provision of advice to parents and guardians as to suitable employment and management.

(*b*) The exploration of facilities for training in crafts, and of the practicability of employment by firms which regularly employ home workers.

(*c*) The provision of accommodation for epileptics during holidays and convalescence.

(*d*) The question of special legislation to include epileptics in the categories of handicapped persons entitled to special care by the State, such as the blind obtain under the Blind Persons Act.

In regard to employment the Council of the Association has been coöperating with the council for the Promotion of Occupational Industries amongst the Physically Handicapped, which already has a number of centres and home teachers for craft training and employment. Warm support has been forthcoming from local authorities, hospitals, and others in the areas in which inquiries have been made, and it remains only to use the experience gained in these localities for the benefit of epileptics the country over.

I should wish in conclusion to pay tribute to the admirable way in which this work has been carried out by Miss Cullen and Miss Craggs, who were responsible both for the actual investigations and for drawing up the reports.

ST. MARY'S HOSPITAL.—Mr. E. W. Meyerstein has given £32,576 to this hospital, which is the price of the freehold of a site of about 10,500 square feet adjoining it in Praed-street. The site will be used for extensions to the hospital.

THE DIONNE QUINTUPLETS

THE Dionne quintuplets were born on May 28th, 1934, and are unique in having reached their present age as an unbroken set. They live close to the farm where they were born, in a self-contained wooden house on an estate of seven acres surrounded by a high metal fence. They are cared for by a staff consisting of two nurses, a teacher, three policemen, a housekeeper and two maids, and four honorary guardians appointed by the Crown attend to their business affairs and to the administration of the considerable income which they derive from motion and still picture contracts and from the endorsement of food products and so on. At birth their average weight was 2 lb. 11 oz. and their medical attendants, Drs. A. R. and W. A. Dafoe, report that at three years they had reached 30 lb. 8 oz.—rather above the normal weight for their age (*Canad. med. Ass. J.* 1937, **37**, 415). In other ways their development has been satisfactory, though the eruption of their teeth has been slow and mentally they are slightly behind the average.

Until the age of five months they were given breast-milk with tomato juice, cod-liver oil and ferrous chloride; evaporated milk with Dextri-maltose and cereal were then substituted. Since they were a year old they have had a varied diet, and gastro-intestinal upsets and feeding difficulties generally have been absent. Although the public have been allowed to view them only through a glass screen and their attendants have worn masks and gowns, they have had occasional mild nasopharyngeal infections. Otherwise they have been healthy, and at three years their only defects were tonsillar hypertrophy and some trivial eye abnormalities.

MacArthur and Ford, quoted by Prof. W. E. Blatz (*Ibid*, p. 424), consider that these quintuplets were derived from a single ovum, for there appears to have been but one placenta and one chorion, and they show remarkable similarity in such characteristics as blood groups, finger-print patterns, facial appearance, hair and eye colour, and the possession of a mild degree of syndactyly. Despite their identical environment, however, conspicuous differences in their social behaviour and compliance with discipline have already appeared. The children are said to be less than usually quarrelsome, and so far there appear to have been no ill effects from the publicity in which they have been brought up.

GEODESY AND CILIARY ACTION

Snow Miller, in his recent book,[1] describes the arrangement of the smooth muscle in the walls of the bronchi and bronchioli as conforming to a "geodesic net-work." A geodesic line on any surface, by definition, is one which traverses the shortest distance between any two points on that surface. On a plane it is clearly a straight line, whereas in relation to a sphere or a cylinder, such as the bronchial wall, it is an arc of a circle and the interlacing of a series of such arcs will produce a geodesic net-work. Mathematically, a geodesic pattern is admirably suited for resisting stresses and strains and for producing an equally distributed pressure within the space enclosed.

Barclay, Franklin, and Macbeth,[2] of Oxford, while studying dust excretion from the lungs, observed, by chance, the tendency of the ciliated epithelium of the trachea to carry foreign particles upwards in the form of a spiral, the movement being clockwise when viewed from above. That this behaviour of the ciliated epithelium of the respiratory tract is not universally recognised is clear as two current text-books of physiology contain no mention of it. The authors pursued the question further by injecting indian ink into the lungs of dogs, sheep, and cats immersed in Ringer's fluid. They found that at the

[1] Miller, W. S., The Lung, London, 1937.
[2] Barclay, A. E., Franklin, K. Y., and Macbeth, R. G., *J. Physiol.* 1937, **90**, 347.

junction of the bronchioli to form bronchi and at the bifurcation of the trachea the movement of the cilia is such that the particles are directed to either side without actually invading the edge of the junction. In the trachea both streams adopt the clockwise spiral.

There would seem to be, therefore, in the trachea and bronchi a relationship between structure and function; for if the spiral pathways of numerous particles, each one starting upon its journey of extrusion from a different point in the bronchial tree, could be visualised they would be seen to make, by their intersections, a moving network closely akin to the geodesic pattern which Snow Miller, from his reconstruction models, claims for the smooth muscle of the bronchial wall.

REST IN BED

REFLECTING upon the remedial value of rest in bed, Prof. Adam Patrick wonders whether it may not sometimes be overdone (*Edinb. med. J.* December, 1937, p. 750). Pyrexia is an absolute indication for rest in bed and no admiration need be wasted on the hero who sticks to his post with a temperature of 104° F.; he should be put to bed at once, by force if necessary. But how long should he be kept there? Excessive rest is bad for the skeletal musculature, so rest in bed should be minimal. But what is the minimum? Dr. Patrick thinks that the spirit of the law is more important than the letter, and that in many pyrexial conditions small exertions such as rising to stool are beneficial rather than harmful. Moreover, the axillary temperature, which is appreciably lower than the oral, should be taken as the standard of normal or otherwise. Irrespective of physical signs in the chest, patients with uncomplicated lobar pneumonia may be allowed up 7 days after the crisis. In rheumatic fever the pulse-rate is more important than the temperature and in this condition Dr. Patrick thinks that rest in bed for 3–4 weeks in the case of adults and 6–8 weeks in the case of children is long enough unless the heart is affected. There are many who hold that, in children at any rate, the heart is always affected; Dr. Patrick, however, puts rheumatic carditis in a separate category altogether, and here he advocates rest in bed for anything from 6 to 18 months, which in all conscience should be long enough. Three weeks in bed is probably enough for uncomplicated scarlet fever, and 5 days, after fever has subsided, enough for measles; although the old rule for measles, "in bed till the rash has gone and indoors till the cough has gone," has much to commend it. The enteric group probably requires 3 weeks post-febrile confinement to bed, and diphtheria at least 3 weeks flat and a fourth week sitting up. Patients with myocardial degeneration, according to Dr. Patrick, may be allowed up in a chair for a little after a few weeks of complete rest; he does not hold, as many clinicians do, that they should often be nursed in a chair from the start. In acute nephritis his practice is to let the patient get up after 4 months if there is no œdema even if albuminuria persists. Of nervous diseases he shrewdly observes that upper motor neurone lesions such as disseminated sclerosis, are usually benefited by rest in bed, whereas those involving the posterior columns, such as tabes, are made worse. Hemiplegics should be got up as soon as possible after the "stroke," perhaps in a week or less, and even severe cases of Addisonian anæmia, with hæmoglobin as low as 30 per cent., are often better up. Subjects of chronic peptic ulcer often do well on a weekly day in bed—or (it may be added) a few days extra when there is exacerbation of symptoms such as commonly follows the sudden onset of cold weather. Speaking tentatively, as a physician must when discussing surgical matters, Dr. Patrick favours early post-operative movements and rising as a means of diminishing the risk of thrombosis and embolism. On the same grounds he disapproves of putting patients in the Fowler position after operation.

NEW PREPARATIONS

COW AND GATE (VITAMIN D) MALTED MILK.—This is a food drink prepared from milk and malted barley and wheat, with the addition of a cod-liver oil concentrate providing vitamin D. It is sold as a powder which may be mixed with milk or with water and drunk hot or cold. The makers (Cow and Gate Ltd., Guildford, Surrey) recommend their product as a supplementary food for children, invalids, and old people, as well as for athletes and others undergoing physical or nervous strain. It is attractive to children and easily assimilated, and is put forward as a suitable drink at bedtime for those who are wakeful.

ALLEN AND HANBURYS LTD. (Bethnal Green, London, E.2) have sent us a copy of their "Medical and Dietetic Products Price List" which replaces their former reference book "Medicamenta Recentia." The new volume contains descriptions of their various specialties, together with a list of sera and vaccines prepared at the Lister Institute and a therapeutic index. The illustrations include pictures of the original premises in Plough-court, Lombard-street, where the firm was founded in 1715, and portraits of some of the distinguished men who have directed it.

A calendar issued by J. F. MACFARLAN AND CO. (32, Bethnal Green-road, London), manufacturers of alkaloids, anæsthetics, and other chemicals, is illustrated with the portraits of the four men responsible for the firm's early growth from the business, founded in 1780, of an Edinburgh surgeon-apothecary. Macfarlan himself was a doctor, being president and then treasurer for 30 years of the Royal Medical Society, as well as an original member of the Pharmaceutical Society. He was helped from the technical side by David Rennie Brown and his son. The former is said to have been the first to produce morphine on a commercial scale in this country. The son, also an expert on the constituents of opium, collaborated with Lister in manufacturing his antiseptic dressings and ligatures.

AN ARTICLE WANTED

THE following letter reaches us from the editor of *Health from Herbs* (1, Ashford-road, Hastings): "It is proposed to open our columns this year to the various systems of medicine other than Herbal Medication, for the purpose of stating their case in favour of their particular system. I, therefore, write to ask whether you would care to supply me with an authoritative article in support of the Allopathic system, or, alternatively, to supply me with the name and address of someone else whom I could approach. The article should not exceed 1000 words and should briefly and plainly set forth the principles of your system, its advantages and (if you desire) disadvantages as compared with other systems; remembering that the article is to be written for the public and not for the profession."

CORRIGENDA

ASSESSMENT OF PSYCHIATRIC TREATMENT.—In Dr. Goodman Levy's letter in our last issue the sentence "it would be irrational to test a minor measure in the first instance" should read "it would be *rational*," &c.

AMINO-ACIDS IN MILK.—In the first sentence of our annotation on this subject (Dec. 25th) the word "diluted" should be "undiluted."

GIFTS FOR LONDON HOSPITALS.—The Hospital Saving Association has distributed £114,000 among 120 hospitals in the London area as a special Christmas gift. Hospitals which benefited include the London Hospital (£7786), King's College Hospital (£2439), Charing Cross Hospital (£2020), Middlesex Hospital (£3528), Guy's Hospital (£4625), and St. Bartholomew's Hospital (£5247).

Vacancies

For further information refer to the advertisement columns

Birmingham, Children's Hosp.—Receiving Room Officer, £250. Res. Sen. Cas. H.S., £125. Also First Asst. to Orthopædic Dept., £100.

Birmingham and Midland Eye Hosp.—H.S., at rate of £130. Also Surg. Reg.

Bolingbroke Hosp., Wandsworth Common, S.W.—H.P., at rate of £120.

Bradford, Royal Eye and Ear Hosp.—H.S., £180.

Brighton, Royal Sussex County Hosp.—Cas. H.S., £120.

Bristol Royal Infirmary.—Sen. Cas. H.S. and Sen. Obstet. H.S., at rate of £150 and £100 respectively. Also H.P.'s and H.S.'s, each at rate of £80.

Calcutta, All-India Institute of Hygiene and Public Health.—Prof. of Maternity and Child Welfare, Rs. 450.

Canterbury, Kent and Canterbury Hosp.—H.S. to Spec. Depts., at rate of £125.

Chester City and County.—Deputy M.O.H. and Deputy School M.O., £600.

Cheyne Hosp. for Children, Chelsea, S.W.—Hon. Aural Surgeon to In-patients.

Colchester, Severalls Mental Hosp.—Asst. M.O., £510.

Croydon General Hosp.—Hon. Asst. Physician.

Deal, Victoria Hosp.—Res. M.O., at rate of £150.

Devonport, Prince of Wales's Hosp.—Jun. H.S., at rate of £120.

Doncaster Royal Infirmary.—H.S., at rate of £175.

Durham County Hosp.—H.S., at rate of £150.

Ealing, King Edward Memorial Hosp.—Cas. O. and Deputy Res. M.O., at rate of £225.

East Ham Memorial Hosp., Shrewsbury-road, E.—Res. M.O., at rate of £200.

Elizabeth Garrett Anderson Hosp., Euston-road, N.W.—Hon. First Asst. and Clin. Asst. for Ophth. Clinic.

Epsom, Surrey County Hosp.—Res. Asst. M.O., at rate of £375.

Guildford, Royal Surrey County Hosp.—H.S., at rate of £150.

Guildford, Warren-road Hosp.—Med. Supt., £800.

Hampstead General and N.W. London Hosp., Haverstock Hill, N.W.—H.P., at rate of £100.

Hosp. for Consumption and Diseases of the Chest, Brompton, S.W.—H.P. Also H.P. for Frimley Sanatorium, each at rate of £50.

Hospital for Tropical Diseases, Gordon-street, W.C.—H.P., at rate of £120.

Hosp. of St. John and St. Elizabeth, 60, Grove End-road, N.W.—Res. H.P., at rate of £100.

Ilford, King George Hosp.—Cas. O. and Surg. Reg., £150.

Infants Hosp., Vincent-square, Westminster, S.W.—Res. M.O., £300.

Kidderminster and District General Hosp.—Jun. H.S., £100.

Leeds, Killingbeck Sanatorium.—Sen. Asst. Res. M.O., £425.

Leicester City Isolation Hosp.—Res. M.O., at rate of £300.

Leicester, City Mental Hospital, Humberstone.—A.M.O., £350.

Lincoln County Hosp.—Sen. H.S., at rate of £250.

Liverpool, Fazakerley Sanatorium.—Res. Asst. M.O., £250.

Liverpool, Royal Children's Hosp.—Res. M.O. and Res. Surg. O. for Heswall Branch, each at rate of £120. Also two Res. H.P.'s and two Res. H.S.'s for City Branch, each at rate of £100.

Liverpool School of Tropical Medicine.—Asst. Lecturer in Dept. of Entomology, £400.

Liverpool University.—George Holt Chair of Pathology, £1200.

London Hosp., E.—Surgical First Asst. and Reg., £300.

London Jewish Hosp., Stepney Green, E.—Surg. Reg., 60 guineas.

London University.—University Chair of Surgery at University College Medical School, £2000. Also University Readership in Medical Parasitology at London School of Hygiene, £900.

Manchester, Booth Hall for Children.—Res. Asst. M.O., at rate of £200.

Manchester, Crumpsall Hosp.—Res. Asst. M.O., at rate of £200.

Morecambe and Heysham Borough.—M.O.H., £800.

Morley Borough.—Asst. M.O.H. and Asst. School M.O., £500.

National Temperance Hosp., Hampstead-road, N.W.—Hon. Anæsthetist. Also Med. Reg., 40 gns.

Norwich, Norfolk, and Norwich Hosp.—H.S. to Special Depts. Also General H.S., at rate of £160 and £120 respectively.

Peterborough and District Memorial Hosp.—Pathologist, £500.

Plymouth, Prince of Wales's Hosp., Greenbank-road.—H.S., at rate of £120.

Prince of Wales's General Hosp., N.—Hon. Clin. Asst. to Children's Dept.

Princess Louise Kensington Hosp. for Children, St. Quintin-avenue, W.—Clin. Asst. for Out-patient Dept.

Reading, Royal Berkshire Hosp.—Cas. O., at rate of £150.

Rochester, St. Bartholomew's Hosp.—Cas. O., at rate of £150.

Royal Air Force.—Temporary Civilian Dental Surgeons.

Royal Cancer Hosp., Fulham-road, S.W.—Res. M.O., £200.

Royal Free Hosp., Gray's Inn-road, W.C.—Registrar to Ear, Nose and Throat Dept.

Royal London Ophthalmic Hosp., City-road, E.C.—Registrar, £200.

Royal Naval Dental Service.—Dental Officers.

Royal Naval Medical Service.—M.O.'s.

St. Albans, Hill End Hosp. and Clinic, &c.—Asst. Med. Director, £500.

St. Bartholomew's Hosp., E.C.—Whole-time Chief Asst. in X-Ray Diagnostic Dept., at rate of £400–£500.

St. Marylebone and Western General Dispensary, 48, Cosway-street, N.W.—Hon. M.O. of Infant Consultations and Children's Dept.

St. Thomas's Hosp., S.E.—Asst. Physician to Dept. of Psychological Medicine.

Salford Royal Hosp.—H.S., at rate of £125.

Sheffield Royal Infirmary.—Sen. Cas. O., at rate of £150.

Stafford, Coton Hill Mental Hosp.—Asst. M.O., £400.

Sunderland Royal Infirmary.—Jun. H.S., £120.

Victoria Hosp. for Children, Tite-street, Chelsea, S.W.—Ophth. Surgeon, H.P. and H.S., each at rate of £100. Also Out-patient Anæsthetist, 10*s.* 6*d.* per attendance.

Walsall Manor Hosp.—Asst. M.O., £350.

Warwickshire and Coventry Mental Hosp., Hatton.—Temp. M.O., £350.

West Bromwich, Hallam Hosp.—H.P., at rate of £200.

West End Hosp. for Nervous Diseases, 73, Welbeck-street, W.—Hon. Med. Psychologists. Two Res. H.P.'s, each £125. Also Registrar to Out-patient Dept., £200.

West Ham Mental Hosp., Goodmayes.—Two Jun. Asst. M.O.'s, each £350.

West London Hosp., Hammersmith-road, W.—Hon. Ophth. Surg.

Wolverhampton and Midland Counties Eye Infirmary.—H.S., £150.

Worthing Hosp.—H.S., at rate of £130.

Medical Diary

January 3rd to January 8th

SOCIETIES

ROYAL SOCIETY OF MEDICINE, 1, Wimpole-street, W.

TUESDAY.

Orthopædics. 5.30 P.M. (Cases at 4.30 P.M.)
Mr. D. McCrae Aitken: 1. Fragilitas Ossium. 2. Dystrophy of Bone. Mr. E. P. Brockman: 3. Fracture of Ankle with Spina Bifida. 4. Myositis Ossificans of Adductors. 5. Traumatic Periostitis of the Tibia. 6. Report on Pathological Fracture of Humerus. Mr. C. Lambrinudi: 7. Metatarsus Elevatus. Miss E. H. Jebens: 8. Pellegrini-Stieda Disease. Mr. Norman Capener: 9. Ossification in the Quadriceps Muscle following Injury. 10. Fracture Dislocation of the Ankle. 11. Giant Cell Tumour, Upper End of Tibia: X Rays, to report progress. 12. Giant Cell Tumour at Lower End of Femur. 13. Subperiosteal Incarceration of Fractured Capitellum.

WEDNESDAY.

Surgery. 8.30 P.M.
Mr. James F. Philip: The Circulation in Gangrene of the Lower Extremities. Dr. Paul Wood: The Question of Cardiac Complications in Major Abdominal Surgery. Dr. R. W. B. Ellis will show a film on Blood Transfusion at the Front by Dr. Frederic Duran-Jordá.

KENSINGTON MEDICAL SOCIETY.

WEDNESDAY.—8.30 P.M. (St. Mary Abbots Hospital, Marloes-road, W.), discussion on Gastro-enteritis in Children.

WEST LONDON MEDICO-CHIRURGICAL SOCIETY.

FRIDAY.—8.30 P.M. (De Vere Hotel), Mr. Eardley Holland, Mr. Kenneth Walker, Prof. V. B. Green-Armytage, and Mr. F. J. McCann: Sterility.

LECTURES, ADDRESSES, DEMONSTRATIONS, &c.

BRITISH POSTGRADUATE MEDICAL SCHOOL, Ducane-road, W.

TUESDAY.—4.30 P.M., Prof. Arthur Ellis: Nephritis.

WEDNESDAY.—Noon, clinical and pathological conference (medical). 3 P.M., clinical and pathological conference (surgical). 4.30 P.M., Dr. Cuthbert Dukes: Surgical Pathology of Diseases of the Rectum.

THURSDAY.—2.15 P.M., Dr. Duncan White: Radiological Demonstration. 3.30 P.M., Mr. Leslie Williams: Disproportion and Difficult Labour.

FRIDAY.—2 P.M., clinical and pathological conference (obstetrics and gynæcology). 2.30 P.M., Mr. C. Naunton Morgan: Diseases of the Rectum.

Daily, 10 A.M. to 4 P.M., medical clinics, surgical clinics and operations, obstetrical and gynæcological clinics and operations.

LONDON SCHOOL OF DERMATOLOGY, 5, Lisle-street, W.C.

TUESDAY.—5 P.M., Dr. A. C. Roxburgh: Syphilis.

THURSDAY.—5 P.M., Dr. J. M. H. MacLeod: Ringworm Infections. (Chesterfield lectures.)

CENTRAL LONDON THROAT, NOSE, AND EAR HOSPITAL, Gray's Inn-road, W.C.

FRIDAY.—4 P.M., Mr. N. Asherson: Pyrexia of Ear, Nose, and Throat Origin.

FELLOWSHIP OF MEDICINE AND POST-GRADUATE MEDICAL ASSOCIATION, 1, Wimpole-street, W.

MONDAY to SATURDAY.—ST. JOHN'S HOSPITAL, 5, Lisle-street, W.C., afternoon course in dermatology (open to non-members).—WEST END HOSPITAL FOR NERVOUS DISEASES, Welbeck-street, W., 8.30 P.M., demonstration on fundus oculi.—PRESTON HALL, near Maidstone, Kent, Sat., demonstration on pulmonary tuberculosis.—Unless otherwise stated, courses are open only to members.